THE CANADIAN ANAESTHETISTS' SOCIETY JOURNAL

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JOURNAL DE LA SOCIÉTÉ CANADIENNE DES ANESTHÉSISTES

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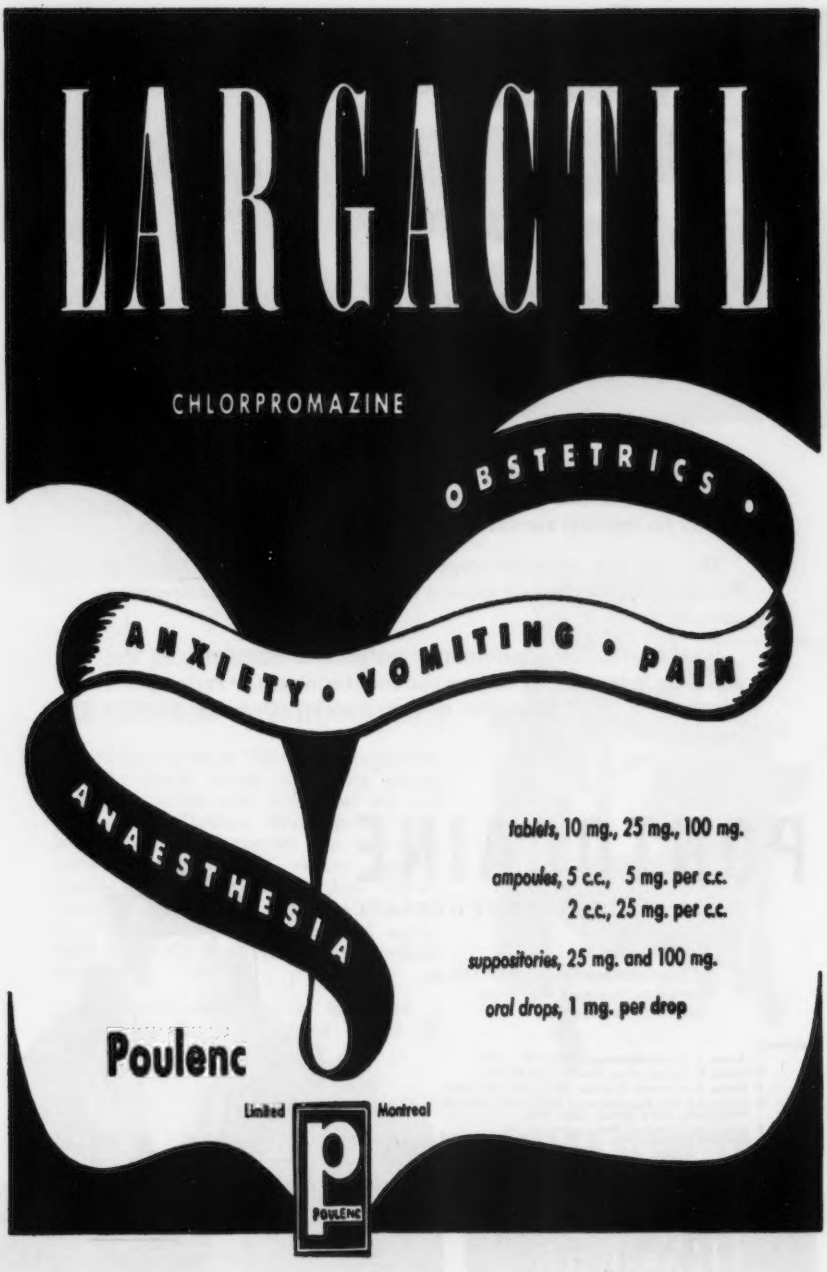
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References:

1. Bonica, J. J.: *Anesthesiology*, 11:716, Nov., 1950.
2. Moore, D. C.: *J. A. M. A.*, 146:803, June 30, 1951.
3. Bonica, J. J.: *Anesth. & Analg.*, 30:76, Mar.-Apr., 1951.
4. Bonica, J.J.: The Management of Pain. Philadelphia, Lea & Febiger, Sept., 1953.
5. Moore, D.C.: Regional Block. Springfield, Ill., Charles C. Thomas, 1953.

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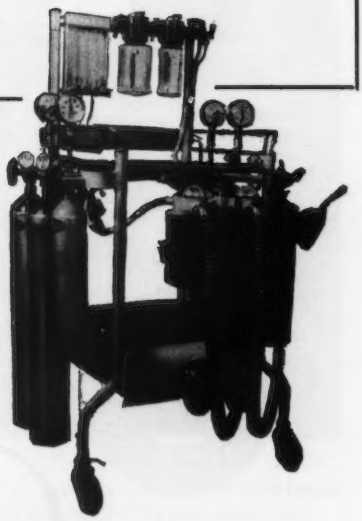

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1. Webster, C.W., C.M.A.J. Oct. 1953.

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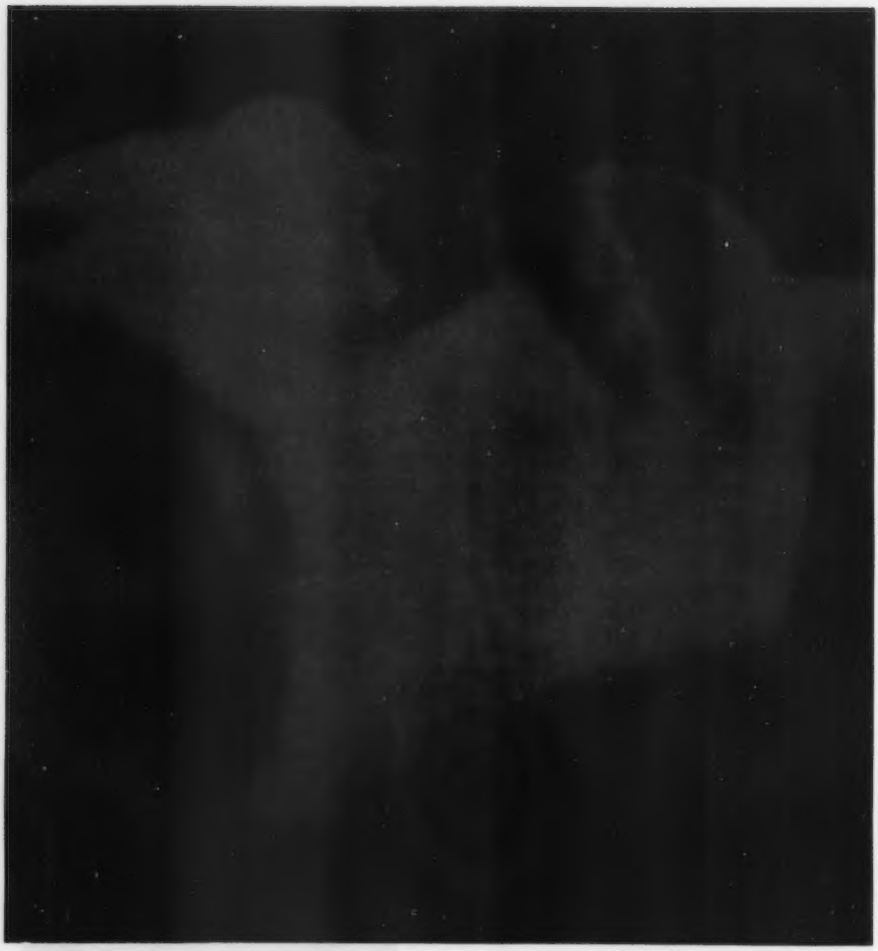

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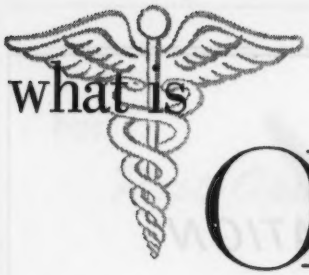

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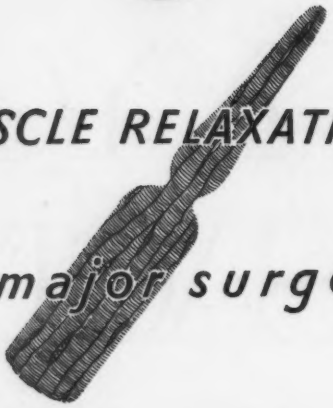

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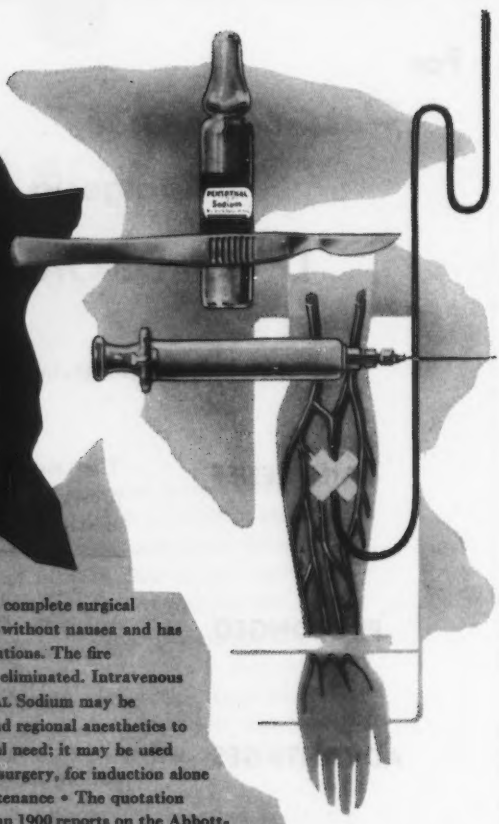

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THE CANADIAN ANAESTHETISTS' SOCIETY JOURNAL

Combining

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Address communications to: The Secretary, Canadian Anaesthetists' Society, 516 Medical Arts Building, 170 St. George Street Toronto 5, Canada

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EDITORIAL

We believe that one of the functions of this Journal should be the publication from time to time of brief review papers covering subjects in the basic sciences and the basic problems of anaesthesia. Such papers have appeared from time to time, but it is the hope of the Editor that this will become a permanent feature of each issue of the Journal. The papers published in this issue by Dr. Robinson and Dr. Keenleyside fall into this group and will, we hope, be valuable particularly to the occasional anaesthetist wishing to refresh his knowledge of the subjects with which they deal.

EDITORIAL

Nous avons l'impression que l'un des principaux buts de ce journal est de publier, de temps en temps, des articles traitant soit de sujets de sciences de base soit de problèmes fondamentaux de l'anesthésie. De semblables articles ont été publiés de temps en temps, mais l'Editeur nourrit l'espoir que ce geste va se répéter à chacun des nouveaux numéros de ce Journal. Les articles que publient, dans ce numéro, les Docteurs Robinson et Keenleyside se rangent dans cette catégorie et ils seront, nous osons l'espérer, une source précieuse de renseignements particulièrement pour l'anesthésiste d'occasion qui cherche à rafraîchir ses connaissances sur les sujets traités.

Honour to Dr. Harold Griffith

We wish here to take note of the award to Dr. Harold Griffith of the Henry Hill Hickman Medal by the Royal Society of Medicine, and to extend to Dr. Griffith our felicitations on the receipt of this important honour. Canadian anaesthetists will, we are sure, take some personal pride in this well-deserved recognition abroad of the work of our distinguished friend and compatriot.

A REPORT ON CANADIAN ANAESTHESIA AND THE CANADIAN ANAESTHETISTS' SOCIETY*

R. A. GORDON, B.SC., M.D., F.R.C.P.(C), F.F.A.R.C.S., D.A.**

As Dr. Harry Shields pointed out in a recent communication to this Society, the first accounts of the practice of Anaesthesia in Canada appeared in the medical literature in June of 1847. In spite of this early beginning, and the notable contributions of Canadian anaesthetists such as Webster, Johnston, Brown, Shields, Bourne, there were in Canada before 1940 not more than half a dozen anaesthetists who were able, economically, to confine their private practice to Anaesthesia. It is my purpose today to draw your attention to the great change which has come over the land since that time, to discuss the factors which have produced this change, and to underscore lightly the part played by the Canadian Anaesthetists' Society in this transition. I shall then, if time permits, take a brief look at the yet obscure outlines of the future, and, perhaps, suggest where our dangers and our opportunities may lie, both professionally and economically.

When I commenced the study of Anaesthesia nearly twenty years ago now, a resident anaesthetist was a novelty in a Canadian teaching hospital, and the specialty was so little regarded by the rest of the profession that a senior surgeon of that time in Toronto, on being informed that one of my contemporaries was applying for a House appointment in Anaesthesia, reacted by saying, "But he was a good student! What on earth does he want to do that for?"

At the time I did my anaesthetic training it never occurred to me that I would be able to earn a living by the practice of Anaesthesia. And then came the opportunity which so many of us shared, of doing full-time anaesthesia in the Armed Forces between 1939 and 1946. We found that the specialty could provide a full-time all-absorbing life-work, with innumerable problems to be solved and an endless chain of questions to which we must find answers.

During the past decade the whole picture of Anaesthesia in Canada has changed. The part-time anaesthetists are now greatly outnumbered by those who are making the specialty of Anaesthesia a full-time life-work. The techniques of Anaesthesia have changed as new problems have been presented, and as older problems have been given newer solutions. The professional stature of the anaesthetist has increased, and his economic status has improved. There is no longer any problem in earning a living from the practice of Anaesthesia, and indeed the shortage of qualified anaesthetists has become an acute problem across the country.

I believe that all these changes have been beneficial, not only to anaesthetists, but to the profession of Medicine as a whole, and to the patients whom we serve.

*An address to the meeting of the Western Divisions of the Canadian Anaesthetists' Society at Vancouver, B.C., April 7, 1956.

**Secretary-Treasurer, Canadian Anaesthetists' Society.

In all of these developments the Canadian Anaesthetists' Society has played an important part.

The Canadian Anaesthetists' Society in its present form was incorporated in 1943, the initiative being taken by the Montreal group, with the co-operation of the senior anaesthetists of Toronto. When I became Secretary ten years ago the membership was 300. Today it stands at 675. The formation of the Society in 1943 was, I believe, prompted by two developments: (1) The suggestion that the Government of the Dominion was about to introduce a Health Insurance scheme, and (2) The institution of Certification in the Specialties by the Royal College of Physicians and Surgeons of Canada.

In the original plans, Anaesthesia was not included as a Specialty for Certification by the Royal College of Physicians and Surgeons of Canada. It was only after most vigorous representations to the College by the Executive Officers of the Canadian Anaesthetists' Society that it was added to the original list. This, I firmly believe, preserved the status of Anaesthesia as a recognized Specialty, and provided the basis for the increase in professional stature which has followed.

When the Royal College of Physicians and Surgeons undertook to certificate qualified specialists in the profession, certification in Medicine and Surgery were included, and were given a lesser status than Fellowship in the College. This, then, created two classes of qualification. Modified examinations for Fellowship were provided for some specialties, but others, including Anaesthesia, were left in the position of having available only the lower grade of qualification. It was evident that this situation was dangerous both professionally and politically, and in 1951 the Society was instrumental through negotiation with the College in having Anaesthesia added to the list of Specialties in which Fellowship in the Royal College of Physicians and Surgeons would be available through a modified examination. The fact that some anaesthetists are Fellows of the College, and that not only have they been able to pass the examination, but that the pass-rate has been good amongst candidates in Anaesthesia, has done a great deal to improve the professional status of anaesthetists in general in the eyes of surgeons and internists. This has economic and political importance in addition to the obvious professional advantage.

One of the early efforts of the Canadian Anaesthetists' Society was towards the establishment of a tariff of adequate fees for the services of the anaesthetist. Such a tariff was required as a guide for the individual anaesthetist, and for purpose of negotiation with economics and tariff committees within the profession, and insurance carriers and agencies of government outside the profession. Such a tariff was maintained until about 18 months ago, with several revisions during the period from 1945 on, and served its purpose very well. I believe that at the present time we are in a somewhat precarious position in this respect, in having no agreed yardstick for negotiations on a national scale.

Another of the functions of the Canadian Anaesthetists' Society has been to deal on behalf of the anaesthetists with the manufacturers of drugs, equipment, and other supplies. We have found manufacturers most anxious to co-operate with us.

Of the problems with which we are still concerned, none has been more

serious and difficult than that presented by the anaesthetist who is the hired servant of a hospital. This problem has been resolved completely except in the City of Montreal. Nothing could be more serious when the day comes that we must discuss the details of a state-sponsored Medical Care plan with agencies of government than to have the employee-anaesthetist still in hospitals in this country. I do not believe that there is need to enlarge on this theme in the City of Vancouver.

Before going on to take a look into the future, there are one or two other things about the activities of the Canadian Anaesthetists' Society which I should like to mention.

The membership policy of the Society, as formulated over the years by Council, has occasionally been criticized. There are those who have wished to use membership in the Society as some sort of professional qualification, and who believe that only qualified full-time anaesthetists should be admitted. The Council, on the other hand, realizing that a large proportion of anaesthetic practice will continue to be done by General Practitioners in smaller communities, has taken the stand that such practitioners should be encouraged to be members of the Society, so that the Society may have the opportunity of cultivating their interest in good anaesthesia, and offer assistance with education to improve their practice.

The *Canadian Anaesthetists' Society Journal* is part of this educational programme, while at the same time it serves as a medium of communication between the anaesthetists of Canada, and brings Canadian Anaesthesia to the notice of the world at large. As your Editor and Manager of the Journal, I can assure you that it has already achieved a place high in the ranks of anaesthetic journals. Today it is distributed on subscription to twenty-five countries outside of Canada, and references to it are becoming frequent in other publications. The Society's other media of education and communication are the Annual Meeting, Regional Meetings such as this present one, and Divisional meetings.

One of the most serious situations in Canadian Anaesthesia today is the continuing shortage of trained anaesthetists. The present rate of hospital construction throughout the country and the inevitable attrition due to death and retirements makes it inevitable that the demand for more qualified anaesthetists will continue at least for some years to come. It may be instructive to examine some of the probable reasons for this disparity between the supply of anaesthetists and the demand for their services, and to determine, if possible, in what measure this situation might be improved.

The present shortage of anaesthetists in Canada undoubtedly had its beginning during the economic depression of the 1930's, when growth of hospitals was largely arrested, and the difficulties of earning a reasonable living from practice effectively blocked the appointment of new members to organized hospital staffs. It was only after the war of 1939–1945 that appointments to established hospitals recommenced, and even then there was little appreciation generally of the possibility of increased demand which the rapid post-war growth of population and of the national economy was to produce. In these early post-war years the number of trainees in Canadian centres was, in fact, reduced below the optimum number by a fear that we were in danger of training more anaesthetists than

would be reasonably employed in Canada. As a result, a number of ex-service personnel who contemplated training in the specialty were lost to it, or moved to the United States and remained there.

Now that we have a better appreciation of the probable demand for anaesthetists, we find that our training facilities are still far from being over-taxed. Why do so few of our recent graduates turn to Anaesthesia as a career? There are undoubtedly a variety of reasons which we should examine with some care.

The first of these reasons is that those of us who have the privilege of contact with the undergraduate student and the junior interne fail to interest them in the specialty of Anaesthesia. This failure on our part is compounded, unfortunately, by a small and dying group of thoughtless surgeons with the "Jehovah" complex who overtly or openly pass on to the student or interne the feeling that the anaesthetist is a lowly person of no consequence, with no opinion worth listening to—a technician to be pushed about and to be made the butt of bad jokes. While it is usually true that these same surgeons demand miracles from the anaesthetists and appreciate their services, this is not a situation likely to recommend the specialty of Anaesthesia as a career to an ambitious young man.

The other important factors which deter many young physicians from embarking on a career in Anaesthesia are the nature of the life they must lead in the specialty, and the lower financial returns they may expect from their post-graduate years in comparison with some of the other specialties.

We have set up in Canada, and rightly so, we believe, high standards of training and qualification in the specialties within the profession. We believe that many individuals undertake this additional training in the hope that they may lead more comfortable lives or gain additional financial rewards as a result of these extra years of discipline in their chosen field. Unfortunately, at present the anaesthetist is likely to be disappointed in both these expectations. Certainly if he has accepted responsibility to provide anaesthesia for patients in a particular hospital, and usually he must, he will find that he is less the master of his own time than is his classmate who went out into the world to do General Practice on the day he finished his junior interneship. While he will, today, probably earn a reasonable living from the commencement of his practice, and reach his peak earnings earlier than his colleagues in some of the other specialties, he will find that his income is unlikely to be better than that of his general practitioner contemporary in the same locality. It is evident that, unless he has a supreme interest in the specialty, the young physician must in these circumstances consider very carefully whether he should invest an additional four or five years of his life to become qualified in this specialty.

I believe that the first step in solving the problem of the shortage of anaesthetists in Canada must be taken by the teachers of Anaesthesia and Surgery in the medical schools. These individuals by arousing in their students and internes a real interest in the problems of Anaesthesia have it in their power to bridge the gap. In this the interest of the surgeon should equal that of the anaesthetist. The problems of remuneration and a reasonable personal life are, we believe, soluble by the intelligent co-operation and planning of those presently practising the specialty with the profession as a whole.

At the present time several major problems appear on the horizon of the future. The certain advent of Health Insurance in some form on a national scale makes it imperative that we should give immediate attention to an analysis of the economic situation of Anaesthesia in relation to the rest of the profession and the economy as a whole, and that we should mend our fences in organizational matters. Nothing is more evident to me, from what I know of the practice of the specialty across the country, than that the anaesthetist is selling his services too cheaply to society in relation to the national economy as a whole. This is leading to deterioration in practice and overwork, for which both the public and the profession will pay dearly unless corrective action is taken soon.

The present situation of university departments of Anaesthesia merits some attention from this Society. The professional standards and reputation of the specialty will, in the final analysis, rest on the standing and competence of the departments in the universities. These departments are, today, seriously in need of financial assistance which will permit at least a small number of people the time to do the thinking, planning, investigation, and teaching which can now be done in too small a way and only at great sacrifice of personal life.

It is evident that it is probably more important in the specialty of Anaesthesia than in most other branches of medicine that provision be made for a retirement income after termination of hospital appointments. We are all aware that in Canada at the present time the provision of such income by self-employed persons is made difficult by discrimination in the income tax act which imposes financial handicaps not experienced by employed persons. The Society is presently exploring methods of overcoming this handicap.

I have attempted here to present to you the general pattern of Anaesthesia in Canada today. I appreciate only too well that the limits of time have made it impossible to deal at all adequately with any single feature of the picture. The details are not well done, but I hope that if you step back and view the picture as a whole, it will all come more or less into focus. We hope that the details may become better defined as we continue to work on the design.

EDITORIAL

SITUATION DE L'ANESTHESIE AU CANADA ET CONSIDERATIONS SUR LA SOCIETE CANADIENNE DES ANESTHESISTES*

R. A. GORDON, B.SC., M.D., F.R.C.P.(C), F.F.A.R.C.S., D.A.**

RÉCEMMENT, à l'occasion d'une réunion des membres de cette société, le Docteur Harry Shields rapportait que les premières traces de la pratique de l'anesthésie au Canada sont apparues dans la littérature médicale en juin 1847. En dépit de ce début précoce et malgré les contributions importantes des anesthésistes canadiens tels que Webster, Johnston, Brown, Shields, Bourne, il n'y avait pas, au Canada avant 1940, plus d'une demi-douzaine d'anesthésistes qui pouvaient, économiquement parlant, se limiter à leur clientèle privée en anesthésie. Je voudrais attirer votre attention, aujourd'hui, sur les changements importants qui se sont opérés depuis ce temps dans notre pays, mentionner les facteurs qui ont produit ces changements et enfin faire la part qu'a jouée la Société Canadienne des Anesthésistes au cours de cette transition. Puis, si le temps le permet, je jetterai un bref regard sur l'avenir encore incertain et peut-être, je désignerai où peuvent se rencontrer nos échecs et nos aubaines, aussi bien du point de vue professionnel que du point de vue économique.

Quand j'ai commencé à étudier l'anesthésie, il y a presque 20 ans maintenant, c'était une nouveauté, dans un hôpital canadien enseignant, que d'avoir un résident en anesthésie. La spécialité d'anesthésie était tellement peu considérée par le reste de la profession qu'un chirurgien senior de ce temps-là, à Toronto, ayant appris qu'un de mes confrères avait fait application pour un stage en anesthésie, s'était exclamé: "Pourtant c'était un bon étudiant; pouvez-vous me dire, dans le monde, pourquoi il fait cela?"

Durant mon entraînement en anesthésie, il ne m'est jamais venu à l'esprit que je pourrais gagner ma subsistance en pratiquant l'anesthésie. C'est alors qu'est arrivée l'opportunité que tant de nous ont exploitée: faire de l'anesthésie à temps complet dans l'armée entre 1939 et 1946. Nous avons découvert que la spécialité pouvait absorber le travail de toute une vie, à temps complet, qu'elle posait d'innombrables problèmes à résoudre et une chaîne interminable de questions auxquelles il nous faut trouver des réponses.

Durant la dernière décade, toute la face de l'anesthésie, au Canada, a changé. Le nombre des anesthésistes à temps partiel est grandement dépassé aujourd'hui par le nombre de ceux qui font de la spécialité leur occupation à temps complet. Les techniques d'anesthésie ont été modifiées à mesure que de nouveaux problèmes sont apparus, puis les problèmes anciens ont reçu de nouvelles solutions. Le status professionnel de l'anesthésiste a pris du prestige et sa condition économique s'est améliorée. Ce n'est plus un problème aujourd'hui que de pouvoir

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**Secrétaire-trésorier de la Société Canadienne des Anesthésistes.

gagner sa vie en anesthésie, alors qu'il manque des anesthésistes qualifiés au point que cela devient un problème aigu dans le pays.

Je suis d'avis que cette évolution s'est faite pour le plus grand bien, non seulement des anesthésistes, mais aussi pour le plus grand bien de toute la profession médicale et des malades au service de qui nous demeurons. A chacun des stades de cette évolution, la Société Canadienne des Anesthésistes a joué un rôle important.

La Société Canadienne des Anesthésistes, sous sa forme actuelle, a été incorporée en 1943; cette initiative a été prise par le groupe de Montréal avec la coopération des anesthésistes seniors de Toronto. Lorsque, il y a 10 ans, je suis devenu secrétaire, le nombre des membres était de 300. Aujourd'hui, il a atteint 675. La formation de la Société en 1943 a été hâtée, à mon avis, à cause de deux éventualités: la première, la rumeur que le gouvernement fédéral était en imminence d'installer un projet d'assurance-santé; la deuxième, l'acceptation de la certification dans les spécialités par le Collège Royal des Médecins et Chirurgiens du Canada.

Au début, l'anesthésie n'était pas au nombre des spécialités acceptées pour la certification par le Collège Royal. Ce n'est qu'après de vigoureux plaidoyers par les membres de l'exécutif de la Société Canadienne des Anesthésistes auprès du Collège que cette spécialité a été ajoutée à la liste originale. J'ai la ferme conviction que ce geste a protégé le status de l'anesthésie en tant que spécialité reconnue, et il a fourni la base pour l'essor de son status professionnel qui a suivi.

Quand le Collège Royal des Médecins et Chirurgiens a entrepris de certifier les spécialistes qualifiés de la profession, la certification en médecine et en chirurgie était comprise, et il leur fut donné un status inférieur à celui d'Associé du Collège. Alors cela a entraîné deux sortes de qualification. Pour quelques spécialités, on a modifié les examens pour le titre d'Associé; pour d'autres spécialités, dont l'anesthésie, on a laissé, comme qualification possible, seulement le degré inférieur. Il était manifeste que cette situation, professionnellement et politiquement, était dangereuse et, en 1951, la Société a entrepris des négociations avec les autorités du Collège pour que l'anesthésie soit ajoutée à la liste des spécialités où le titre d'Associé du Collège Royal des Médecins et Chirurgiens pourrait être obtenu par des examens spéciaux. Le fait que quelques anesthésistes soient Associés du Collège et que, non seulement ils aient réussi à passer l'examen, mais qu'en plus, parmi les anesthésistes, le pourcentage des candidats heureux soit bon, ce fait a contribué grandement à améliorer le status professionnel des anesthésistes en général aux yeux des chirurgiens et des internistes. Cela donne un prestige économique et politique en plus de l'avantage professionnel.

Un des premiers efforts de la Société Canadienne des Anesthésistes a été de fixer une échelle d'honoraires adéquats pour les services de l'anesthésiste. Une telle échelle s'est avérée nécessaire comme guide pour l'anesthésiste lui-même, pour discuter, au sein de la profession, des ententes tarifaires avec les différents comités économiques et, en dehors de la profession, avec les porteurs d'assurance de même qu'avec les agences gouvernementales. Une échelle de ce genre a été en force jusqu'à il y a environ 18 mois en subissant de nombreuses modifications de 1945 à nos jours et elle s'est avérée très pratique. J'ai l'impression que, actuellement, nous sommes dans une situation assez précaire en cette matière, parce

que nous n'avons pas accepté de barême pour négocier sur une échelle nationale.

Une autre des fonctions de la Société Canadienne des Anesthésistes a consisté à discuter, au nom des anesthésistes, avec les manufacturiers de médicaments, d'instruments et de tout autre matériel. Les manufacturiers nous ont assurés de leur plus entière collaboration.

Parmi les problèmes qui nous occupent encore, le plus sérieux et le plus difficile est celui que pose l'anesthésiste employé d'un hôpital et loué en quelque sorte. Ailleurs que dans la Cité de Montréal, nous avons complètement résolu ce problème. Quand il deviendra nécessaire de discuter les détails d'un plan d'assurance-santé payé par l'état avec les agences gouvernementales, ce serait une situation embarrassante que d'avoir encore, dans ce pays, des anesthésistes employés par des hôpitaux. Je ne crois pas qu'il soit nécessaire d'insister sur ce sujet dans la Cité de Vancouver.

Avant de jeter un coup d'œil sur l'avenir, j'aimerais à mentionner un ou deux détails concernant les activités de la Société Canadienne des Anesthésistes.

On a critiqué occasionnellement la politique de recrutement des membres de la Société de la façon décidée par le Conseil depuis des années. Il y a un groupe qui désirerait que l'acceptation comme membre de la Société serve en quelque sorte de qualification professionnelle; ainsi ils préconisent que seulement les anesthésistes qualifiés et à plein temps soient acceptés. Le Conseil, d'autre part, estimant qu'une grande partie de la pratique de l'anesthésie va continuer de se faire, dans les petits centres, par les praticiens généraux, a émit l'opinion que ces praticiens devraient être invités à devenir membres de la Société; en ce faisant, la Société pourrait prendre l'occasion de les stimuler à pratiquer de la bonne anesthésie et, tout en améliorant leurs connaissances, améliorer leur pratique.

Le *Journal de la Société Canadienne des Anesthésistes*, tout en servant de moyen de communication entre les anesthésistes du Canada, fait partie de ce programme éducationnel; de plus, il porte à la connaissance de l'univers entier les nouvelles de l'anesthésie canadienne. En qualité d'éditeur et de gérant, je puis vous assurer que notre journal a déjà atteint une place enviable parmi les journaux traitant de l'anesthésie. Aujourd'hui, il compte des abonnés dans 25 pays à part le Canada et, dans les autres publications, on trouve de plus en plus de références à notre journal. Un autre medium d'enseignement et de communication de la Société, c'est son assemblée annuelle, des assemblées régionales comme celle-ci et les assemblées des divisions.

Aujourd'hui, une des situations les plus sérieuses de la Société est le manque continuel d'anesthésistes entraînés. Le rythme actuel de la construction des hôpitaux à travers tout le pays et l'inévitable tribut payé à la mort et à la retraite font que, à coup sûr, la demande pour des anesthésistes qualifiés va continuer encore au moins pour quelques années à venir. Il peut être intéressant de passer en revue les raisons probables de cet écart entre l'offre et la demande d'anesthésistes et de dire, si possible, jusqu'à quel point cette situation peut être corrigée.

Sans aucun doute, le manque actuel d'anesthésistes au Canada a commencé au cours de la dépression économique de 1930, alors que la construction des hôpitaux a considérablement ralenti et que les difficultés de gagner une existence

convenable en pratique ont bloqué radicalement le désir des nouveaux membres d'organiser les services des hôpitaux. Ce n'est seulement qu'après la guerre 1939–1945 que les applications pour organiser les hôpitaux ont recommencé et, même alors, il était très difficile de prévoir la demande accrue que devaient provoquer l'augmentation rapide de la population et l'essor de l'économie nationale d'après-guerre. Au cours de ces premières années d'après-guerre, le nombre de candidats à l'entraînement dans les centres canadiens était, de fait, inférieur au nombre idéalement requis, parce que nous craignions d'entraîner plus d'anesthésistes qu'il n'y aurait d'emplois au Canada. Comme résultat, un certain nombre de militaires licenciés qui comptaient faire leur entraînement dans la spécialité ont abandonné leur projet ou ils ont émigré aux Etats-Unis et ils y sont demeurés.

Maintenant que nous avons un meilleur aperçu de la demande pour les anesthésistes, nous nous rendons compte que nos facilités d'entraînement sont loin d'être exagérées. Pourquoi un si petit nombre des nouveaux promus se dirigent-ils vers l'anesthésie pour s'y faire une carrière? Il existe sans doute un grand nombre de raisons qu'il conviendrait d'apprécier avec soin.

La première de ces raisons est que ceux d'entre nous qui avons l'occasion de rencontrer les étudiants universitaires et les internes juniors ne nous appliquons pas à les intéresser à la spécialité. Cette lacune de notre part est compliquée, malheureusement, par un petit groupe qui tend à disparaître, groupe de chirurgiens irréfléchis, à complexe de transcendance, qui, ouvertement et couramment, communiquent à l'étudiant ou à l'interne leurs convictions que l'anesthésiste est personnage d'un rang inférieur et sans responsibilité, dont les opinions sont superflues, en somme un technicien à mettre de côté et un objet de plaisanteries. Toutefois, c'est habituellement la vérité, que cette même catégorie de chirurgiens exigent des miracles des anesthésistes et apprécient leurs services: une pareille situation ne constitue pas une recommandation pour la spécialité d'anesthésie comme carrière pour un jeune homme qui a de l'ambition.

Les autres facteurs importants qui empêchent un grand nombre de jeunes médecins de se diriger vers l'anesthésie pour en faire une carrière sont: d'abord la nature du travail à faire et la vie à mener dans la spécialité, ensuite le revenu financier inférieur à attendre de leurs années d'études post-universitaires en comparaison avec ce que peuvent procurer les mêmes années dans d'autres spécialités.

Au Canada, et à bon droit, je crois, nous avons établi des exigences sévères d'entraînement et de qualification pour les spécialités médicales. Nous avons l'impression que plusieurs individus se soumettent à cet entraînement additionnel dans le but de mener une vie plus facile ou de gagner davantage comme résultat de ces années supplémentaires d'étude dans la spécialité choisie. Dans le moment, malheureusement pour l'anesthésiste, ces deux expectatives pourraient bien tourner en déception. A coup sûr, s'il a accepté la responsabilité de donner l'anesthésie aux malades d'un hôpital en particulier et, d'habitude, il doit le faire, il se rend bientôt compte qu'il est encore moins libre que son confrère qui, le dernier jour de son internat terminé, s'est ouvert un bureau en clientèle générale. Aujourd'hui, il est possible qu'il gagne une vie convenable dès le début de son entrée en service, il est aussi possible qu'il atteigne son revenu maximum

plus tôt que ses collègues dans les autres spécialités, mais il va aussi se rendre compte que son revenu, vraisemblablement, ne dépassera pas celui de son confrère en pratique générale dans la même localité. Il devient logique, en ces circonstances, qu'un jeune médecin, à moins qu'il ait un intérêt capital dans la spécialité, doive réfléchir attentivement avant de sacrifier quatre ou cinq années de sa vie pour se qualifier dans cette spécialité.

Je suis d'avis que le premier geste à faire pour essayer de résoudre le problème du manque d'anesthésistes au Canada doit être fait par les professeurs d'anesthésie et de chirurgie des écoles de médecine. En éveillant chez les étudiants et chez les internes un réel intérêt des problèmes de l'anesthésie, ces professeurs ont le poids qui peut faire osciller la balance. En cette matière, l'intérêt du chirurgien devrait être aussi grand que celui de l'anesthésiste. Quant au problème de la rémunération et à celui de faire une vie raisonnable, nous croyons qu'avec la coopération et les projets de ceux qui pratiquent actuellement la spécialité ils sont faciles à résoudre surtout avec une coopération intelligente de toute la profession.

Actuellement, nous voyons poindre à l'horizon de nombreux problèmes d'importance. L'apparition d'un plan d'assurance-santé sur une échelle nationale nous obligerait de faire un relevé immédiat de la situation économique de l'anesthésie comparée au reste de la profession ainsi que de son économie entière et de hâter nos protections organisées. Rien ne me semble plus évident, connaissant bien la pratique de l'anesthésie dans tout le pays, que l'anesthésiste offre ses services à la société à trop bon marché si l'on tient compte de toute l'économie sociale. Cette situation peut tout gâter et conduire au surmenage, ce qui pourrait coûter cher au public et à la profession à moins que des mesures correctrices soient prises très bientôt.

La Société doit s'intéresser à la situation actuelle des services universitaires d'anesthésie. En dernière analyse, le status professionnel et la renommée de la spécialité reposent sur le niveau scientifique et la compétence des services universitaires. Aujourd'hui, ces services ont grand besoin d'appui financier afin de donner à un petit nombre d'individus le temps de penser, de faire des projets, de la recherche, de l'enseignement ce qui, actuellement, ne peut se faire que sur une trop petite échelle et seulement au prix de sacrifices personnels trop considérables.

Il est probablement plus important dans la spécialité d'anesthésie que dans la plupart des autres branches de la médecine qu'il soit possible de prévoir une pension de retraite à la limite d'âge d'emploi dans les hôpitaux. Nous savons tous que, à l'heure actuelle, au Canada, la prévision d'une pension pour les personnes sans employeur est rendue difficile à cause de la loi de l'impôt sur le revenu qui impose à cette catégorie de personnes des sacrifices financiers que ne connaissent pas les personnes ayant des employeurs. Actuellement, la Société essaie de trouver des moyens de surmonter ces difficultés.

J'ai essayé de vous présenter, ici, une vue d'ensemble de l'anesthésie, au Canada, aujourd'hui. Le temps limité m'a empêché de traiter adéquatement aucun des aspects de ce tableau, les détails ne sont pas bien soignés mais je crois que, si vous reculiez pour avoir une vue d'ensemble du tableau, le tout deviendrait plus ou moins au point. Nous sommes persuadés que les détails pourraient être améliorés si nous continuions à travailler sur le tableau.

TWENTY YEARS OF ANAESTHESIA IN THORACIC SURGERY*

GEORGES COUSINEAU, M.D., F.F.A.R.C.S., LEONARD LEGAULT, M.D., and LEO FOREST, M.D.**

THE MAJOR PRINCIPLES of cardio-respiratory physiology have been known to us for a long time. However, many new ideas have been added to the classical principles. Anaesthesiology, a much younger science, has evolved in such a prodigious way in the last twenty years, that few techniques of those early days are being used today. For this reason, it appeared to us that it would be very interesting to review this evolution in a centre specializing in thoracic surgery. The centre in question is the Sacred-Heart Hospital of Montreal, with a bed capacity of 900 (of which 50 beds are allotted to thoracic surgery), which has been functioning for the past twenty-three years. From 1935 to 1955, the authors of this paper have successively directed the Department of Anaesthesia of this hospital. In this paper each author reports for the period when he was director.

DR. GEORGES COUSINEAU, 1935–1945

I shall discuss the Iron Age, from 1935 to 1945. My colleagues will report the evolution in our techniques, its success being indicated by the improved statistics. However, we must keep in mind that the type of patient submitted for thoracic surgery has changed.

Twenty years ago, who did we subject to thoracic surgery? Two kinds of patients:

1. The patients abandoned by all: carriers of nauseating bronchial secretions. The general status of these patients was poor because of the destructive processes in the lungs. Morale was at its lowest. Most patients asked for only one thing, namely, to get the operation over with as soon as possible.

2. The tuberculous patient in the third phase, that is, the tuberculous patient with fibrous cavities. To prepare these patients for surgery, we were limited to postural drainage, coramine, and digitaline. Owing to the presence of abundant secretions, many patients had to be aspirated prior to the surgical procedure. This then gave a partial or temporarily dry bronchial field, but diminished considerably the general resistance of the patient. The technique of bronchoscopic aspiration was very crude as compared with present procedures.

However the major principles of anaesthesiology were sufficiently known for thoracic surgery. Thoractomy is the opening of the pleural cavity which permits the atmospheric air to enter the pleural spaces. Therefore, three factors produce pulmonary collapse, namely, atmospheric pressure, the elasticity of the pulmonary tissue, and the abolition of the surface tension of the pleural spaces. Furthermore, as the equilibrium of the intrathoracic tension is abolished, there is danger of

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**Sacred-Heart Hospital, Montreal, P.Q.

mediastinal shift and paradoxical respiration, producing progressive anoxia and contralateral infection. We must further remember that in the diseased lung the vital capacity is reduced and abundant purulent secretions can spill from the diseased lung to the healthy one as the patient is lying on his healthy side during the operation. The operative technique is not to be neglected as the crudeness of the operation has been the cause of bronchiolar spasm and of nervous reflexes, producing a state of operative shock.

We did understand the importance of assisted pulmonary ventilation, but our technical methods at that time seem elementary today.

There were two schools of thought on the conduct of anaesthesia. (*a*) The first called for a very light anaesthesia which preserved the cough reflex. This permitted the emptying of the bronchial tree but at the same time caused mediastinal shift and diminished vital capacity. High spinal anaesthesia and general anaesthesia with nitrous oxide with the open circuit fall into this category. (*b*) The second method was deep anaesthesia with respirations assisted by the rhythmic compression of the anaesthetic bag. This last school gradually developed the present status of intratracheal anaesthesia. The improvement of the instruments—laryngoscopes, intratracheal tubes—and the greater dexterity acquired by the anaesthetist in introducing intratracheal tubes added to the popularity of this technique.

This was the era of general anaesthesia in a closed circuit. Cyclopropane was making its début. In Montreal, cyclopropane became popular thanks to the researches of Griffith, while in Boston, under the influence of Beecher, ether, nitrous oxide and oxygen, were favoured. This was also the beginning of intravenous anaesthesia with the barbiturate evipal, and we used it in thoracic surgery. As we review these first ten years of anaesthesiology for thoracic surgery, at Cartierville, we see a succession of techniques. These different techniques reflect the efforts of the anaesthetist to maintain the principles of cardio-respiratory physiology. In retrospect, from old records, the first intrathoracic intervention was very elementary. However, today, we respect the courage, the tenacity, and the spirit of research of the pioneers in thoracic surgery.

Today, we stress the team work of the surgeon, internist, and anaesthetist. This also existed twenty years ago. In the early days the team worked together with one thought: the mechanical excision of an organ in record time. From the study of these charts tarnished by time, and containing few details of the techniques employed, it is easy to comprehend the uneasiness and anguish of these workers and their tenacity in following their task even though the results were very discouraging.

The first three intrathoracic interventions date back to 1934. All three were defeats. The surgical technique employed was the tourniquet applied at the pulmonary hilum. The general anaesthesia was done with nitrous oxide—oxygen; an ordinary mask without "rebreathing" was used. The pulse was recorded at three-minute intervals, but the blood pressure was rarely registered. There was no transfusion during the intervention, only an intravenous infusion. After the completion of the operation, a transfusion was given by the direct method. Time of the operation was from one to two and a half hours. Two of these patients awakened,

and were completely conscious one and a half hours after the operation, but unfortunately died the following day, owing to progressive anoxia and cardiac insufficiency. The third patient died on the operating table, forty minutes after the anaesthesia had begun. The surgical technique was identical, but a new anaesthetic method was employed, evipal given intravenously. Cause of death was overdosage with barbiturate, used for the first time in America.

Since 1935, the improvement in statistics has been progressive from year to year. During these 10 years, 1400 patients were submitted to thoracic interventions with a mortality rate of 5 per cent.

We shall now mention two cases of that decade which are of particular interest.

1. *6–11–35*

Yvette B., age 10, underwent a right pneumonectomy for suppurative bronchiectasis. The surgical technique was the tourniquet applied to the pulmonary hilum, followed by complete excision of the lung. The time of operation was 1 hour and 43 minutes. The anaesthetic technique was simple. Nitrous oxide and oxygen was used with an ordinary mask without intratracheal intubation. No transfusion, only intravenous infusion, the pulse rate of 132–150 taken at intervals of 3 to 5 minutes, but no blood pressure recorded. Excellent results, the patient leaving the hospital cured, two months after the intervention.

2. *20–9–40*

Lucette D., age 2, brought to the operating room for right pneumonectomy for multiple pulmonary abscesses. Duration of operation, 2½ hours. The anaesthesia, with intubation under direct vision, was done with cyclopropane and oxygen; blood pressure and pulse were recorded at 5-minute intervals. A transfusion of 100 cc. of citrated blood was given during the operation followed by a second transfusion of 175 cc. The endotracheal tube was borrowed from Dr. Griffith, who was always willing to help improve anaesthesia. The result was excellent; the child left the hospital two and one half months after the intervention, perfectly well.

It is interesting to note the speed of the surgeons: for example, a thoracoplasty first stage took thirty minutes; second stage, twelve minutes. Also certain patients had as many as twelve interventions on the thorax, all of these within eighteen months.

In the years 1937–38, the technique was intratracheal anaesthesia with cyclopropane and oxygen in a closed circuit, the induction being done with evipal, later pentothal. The respirations were often assisted and sometimes completely controlled. And thus gradually we reached a new decade. The work of the anaesthetist has improved as a result of the better understanding of the basic principles, the exchange of views, publications, and the acceptance of the principle of team work.

Dr. Leonard Legault, 1945–1950

After long hesitant years in thoracic surgery, we have now arrived at a stage where success is finally attained with a period of more daring experiments. Working together, the anaesthetist has greatly improved his techniques. We are following in the steps of courageous pioneers who showed us the way to success by their studies and researches. Operations both intra- and extra-thoracic grow

every day in number and quality, and the success obtained is the result of a better co-operation between the surgeon and the anaesthetist.

At present, the techniques most frequently employed are general anaesthesia with pentothal, cyclopropane, nitrous oxide and ether. Intratracheal intubation is now a routine, since the use of curare, which renders it so easy, is now more familiar. Intubation permits suction at any time during the operation and keeps the respiratory tract free of the secretions that may obstruct the respiratory system, and in so doing, almost eliminates the dangers of atelectasis and of spreading infection.

For some time now, thoracic surgery has been improved by bronchoscopy, and as a result the patients who are to be operated on, have had their respiratory tract well prepared, and with far better results than with postural drainage. We must also add that the patients are better selected and have received better pre-operative care. Cardio-respiratory physiology has permitted a more complete study of the patients and, as a result, the thoracic surgeon works under more favourable conditions for himself and the patients.

It was during the period under review that our attention was focused on epidural anaesthesia, which was described by Fugihama, Neves, Brasler and Buckingham in the *Journal of Thoracic Surgery* for February 1948.

The solution employed was 30 to 40 cc. of novocaine and cobefrine 1/2000,000 to which we added 10 to 15 mgm. of pontocaine to produce a longer lasting anaesthesia.

We have used this method for poor risk patients, to avoid the danger of spreading infection, and in patients who had previous thoraco-plastic surgery, and had a reduced cardiac, renal, and respiratory capacity. The results obtained proved without doubt the importance of epidural anaesthesia for certain indicated cases.

Patients operated on for thoracoplasty, 1st stage	26
Patients operated on for thoracoplasty, 2nd and 3rd	82
Perfect anaesthesia without supplementation	70%
Good anaesthesia with pentothal in I.V. solution	25%
Poor anaesthesia with addition of pentothal and nitrous oxide	5%

This series was without a death or serious accident, and we may say that epidural anaesthesia was of great help in the worst cases, since it makes perfect oxygenation possible during the entire operation, and also prevents postoperative atelectesia.

We shall now end this part of the symposium by giving you the general statistics in thoracic surgery for the years 1945 to 1950:

Years	No. of operations	Intra-thoracic	Extra-thoracic	Death	%
1945	300	69	211	15	5
1946	332	105	227	14	4½
1947	356	133	223	13	3.6
1948	385	132	253	10	2.6
1949	300	82	218	5	1.66
1950	407	75	332	6	1.4

You may therefore judge for yourself the progress constantly made in thoracic surgery; without being boastful about it, we believe the anaesthetists were of great help. A few words from Dr. G. A. P. Hurley may perhaps be quoted here: "If thoracic surgery has made so much progress in the last twenty years, it is only with the help of the anaesthesia group, who have made successful those new modern methods of anaesthesia."

Dr. Leo Forest, 1950–1955

Today, the Department of Anaesthesiology of the Sacred-Heart Hospital is getting constant help from a full-time internist. Our department is also working in close co-operation with the department of cardio-respiratory physiology of the Lavoisier Institute. All patients facing a major thoracic operation are first thoroughly investigated by a group of specialists in respiratory functions at this Institute. Following this, a consultation between the surgeon, the anaesthetist and the internist is required before the operation. The method of anaesthesia and the position of the patient on the operating table are decided during this consultation. The internist then takes over for the entire pre- and postoperative period.

Thoracic surgery requires closer supervision on the part of the anaesthetist than any other surgery. Therefore, the technique used must be as simple as possible. In a case of bronchiectasis we use either bronchial intubation with a Carlens' tube or the two-tube method, one in the left bronchus and the other in the right. In a case of external bronchial fistula we use bronchial intubation. Our anaesthetists are trained in bronchoscopy and the aspiration of patients just before intubation.

For patients with emphysema, the orotracheal tube permits light anaesthesia which in turn allows better control of the electrolytic balance. This is very important in all cases of pulmonary emphysema.

For a time we made use of an automatic ventilation unit, but we have discontinued this. It was too difficult to synchronize the system with the spontaneous breathing of the patient.

For induction, we use barbiturates and maintenance is carried out with nitrous oxide, ether, and cyclopropane. Good anaesthesia for thoracic surgery means perfect control over respiration and circulation.

Though unobstructed respiration is important during any kind of operation, it is without any doubt much more important during operations where the lungs and the heart are manipulated by the surgeons' hands. Pulmonary ventilation must be adequate for the maintenance of normal oxygen and carbon dioxide blood levels.

As for circulation, the anaesthetist must always bear in mind that the normal negative thoracic pressure helps greatly the return of blood to the right heart during inspiration. When the thoracic cavity is open, this mechanism does not work any more and the filling of the right heart is slowed. Moreover, the surgeon is manipulating areas of great nervous reflex activity. It is up to the anaesthetist to control, to decrease, or to inhibit completely these reflexes. For this, he may have to use atropine, novocaine, physostigmine, or curare according to

the situation. These drugs are to control the unwanted reflexes of sympathetic or parasympathetic nervous systems.

It is needless to say that most thoracic operations mean considerable blood loss for the patient. We have adopted the system of weighing sponges. Though simple, this system is the most accurate way of figuring how much blood to give the patient during and after the operation.

The noted improvement in the statistics of the department of thoracic surgery shows the necessity of close co-operation in hospital work. The mortality rate of the department is now 0.8 per cent as compared with 6 per cent only a few years ago.

This improvement is the result of better surgical technique, better hospital equipment, and, above all, better anaesthesia because the anaesthetist understands cardio-respiratory physiology.

Summary

The authors have been, in succession, directors of the Department of Anaesthesiology of the Sacred-Heart Hospital of Montreal for the past twenty years. They have combined in this paper to outline the evolution of anaesthesia over this period in the thoracic surgery unit of this hospital.

EVALUATION OF ANALGESIC DRUGS WITH SPECIAL REFERENCE TO SUBJECTIVE RESPONSES*

E. E. DANIEL, PH.D.**

INTRODUCTION

THE EXPERIENCE of pain, like other sensations, is a composite phenomenon. It must involve at least the stimulation of receptors, the transmission of this stimulation to the control nervous system, and the integration of the pain impulses in the CNS with other impulses to produce the response of the organism. Clearly, then, there are several places at which one might interfere with the pain experience. One might prevent the stimulation of receptors by avoiding the stimulus or preventing the response of the receptors, or one might interfere with the transmission of pain or its integration at various levels. Such measures as local anaesthesia, nerve or cord sections, or frontal lobotomies (9) have been used to remove an unbearable pain experience.

Furthermore, as every anaesthetist knows, the total experience of pain is affected by the past experience and training of the organism. Some patients report little or no pain with procedures identical with those producing an unbearable experience in others. In a study of unnarcotized wounded in the last war, Beecher *et al.* (1) could find no correlation between pain and the severity of the wound.

Hill and co-workers (15–18) in a series of papers have shown that, both in man and in animals, anxiety over the anticipation of pain is an important part of the pain experience and leads to disruption of organized behaviour. Furthermore, it is well known that emotion can obviate or intensify the reaction to pain. For example, Isbell (19) has shown that emotional disturbance of post-addicts at Lexington (produced by unexpected searches, etc.) prevented or even reversed in some cases the usual action of morphine on pain threshold.

Clearly then, the pain experience may be modified by training or conditioning of the organism in the past or by social or environmental events in the present.

Therefore, when one comes to analyse the action of drugs which modify the pain experience, it is necessary to consider possible effects on many sites: on the pain receptors, on the transmission paths, on the integration of pain with impulses set up by other stimuli at various levels of the central nervous system, on the process of stimulation of other impulses by agents in the internal or external environment, on the past training of the organism in integrating such sensations.

EVALUATION OF ANALGESICS USING EXPERIMENTAL PAIN THRESHOLDS AND PROPOSED MECHANISM OF DRUG ACTION

The situation seems to have become confused owing to rigid adherence to an artificial classification of the possible sites of drug action. Hardy, Wolff and

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**Department of Pharmacology, University of British Columbia, Vancouver, B.C.

their co-workers (13, 14, 30) proposed that analgesic drugs (e.g., acetyl salicylic acid) act primarily by raising the "pain threshold" while others (e.g., opiates) act not only by this mechanism, but also by decreasing pain intensity and by decreasing the "reaction to pain." In the case of opiates, they add "the promotion of sleep" as part of their analgesic action. Beecher (3) in studies on clinical pain has emphasized the dichotomy between pain sensation and responses to pain to an even greater degree, though he denies that Hardy *et al.* (7) have succeeded in separating threshold for sensation from reaction.

When one attempts to give a material meaning to these proposed mechanisms, some difficulties arise. What threshold is elevated—that of the pain receptors, or of some synapse along the transmission path or of some integrating centre? Further, where does the sensation of pain cease and the reaction to pain begin? (In other words, are our reactions to pain, such as reflex withdrawal or muscle spasm or vocalization, a part of the painful experience?) Is there any interaction between the "sensation" and the "response" or the "anticipation"? (Judging from Isbell's work, repeated painful responses lead to anxiety and to alteration in subsequent painful experiences.) What does sleep do? Does it raise the pain threshold or alter response to pain? Despite this lack of precision as to the meaning of the dichotomy, Hardy *et al.* (12) have claimed that their radiant heat technique yields effects on the sensation of pain more or less independent of the reaction to it. Beecher *et al.* (2, 3) on the other hand, believe that their studies on clinical pain are mainly concerned with reaction to pain.

To begin to straighten out the existing confusion, it seems necessary to go back and re-examine briefly the experimental evidence on which the separation of the sensation of pain from the response to it was based.

Hardy, Wolff and collaborators developed a system using radiant heat to produce repeatable physical changes on the forehead, leading to pain. They showed, using themselves as subjects, that a threshold to this pain could be repeatedly determined with minimal variation on the same day. There was considerable variation from subject to subject and in the same subject from day to day. However, they found that using a more or less standardized procedure acetyl salicylic acid derivatives raised the "pain threshold" some 30–60 per cent, and that narcotics like morphine could elevate the "threshold" over 100 per cent.

How did they know that this was a change in the sensation of rather than the response to pain? They took as a measure of the reaction to pain, the electrodermal response to the pain (a change in skin resistance). This, in contrast to the pain threshold, they found to be variable and affected by emotional states, fatigue, etc. This response was much reduced by opiates, but not by salicylates, even though these also elevated the pain threshold.

Several questions immediately come to mind.

1. Since the determination of pain threshold was subjective, what role does suggestion play? In other words, what is the response if the subject does not know the nature of the drug? In other studies, conducted by techniques which permit evaluation of the role of suggestion (use of placebos, double blind technique), no consistent elevation of pain thresholds by ASA type analgesics compared to placebos could be demonstrated (26) while elevations produced by opiates (26, 19) were much less marked. Subjective responses therefore are

influenced by suggestions (e.g. from taking of a drug) that pain will be reduced and the occurrence of marked pain relief or elevation of threshold in response to placebos bears this out.

Hardy, Wolff and collaborators (14, 32) answered this objection by the claim that only trained subjects can determine thresholds and discriminate between drugs and placebos. In our own laboratory, using two different methods of determining pain threshold (deep pain from a pressure cuff on the lower leg and periosteal pain by point pressure on the forehead (8)), we have confirmed previous failures to find consistent differences between drugs and placebo effects on "threshold" with ASA type compounds and found a significant but minor elevation of 15 per cent only in the case of meperidine. Furthermore, training of some of the subjects over an entire summer did not improve their ability to discriminate between drug and placebo or did not reduce remarkably the variation from various sources in their threshold determinations. Such results leave doubt as to how the relief of pain by drugs clinically in a wide variety of individuals is related to a situation in which change in the pain sensation cannot be consistently demonstrated in the trained subject. Are we to conclude that the only effect of acetyl salicylic acid is that of a more or less expensive placebo, or that our methods are as yet inadequate? Even in the case of narcotics, where significant elevations over placebo thresholds have been found (8, 26), though not invariably (7), there is no proof that the subjects may not have learned to distinguish drug from placebo by some action other than that of pain sensation. For example, in our study a large fraction of the students receiving meperidine reported feeling nausea, or drowsiness, or some other drug effect, not reported with ASA compounds or placebos. In a careful study of this problem, Lasagna *et al.* (21) have shown that narcotics do produce marked subjective dysphoria (not euphoria) in normal healthy individuals. There seems to be a distinct possibility that a subject might learn to recognize mood changes or other effects from opiates and by suggestion and conditioning respond by elevating their pain threshold. Lasagna showed that individuals with such training, post-addicts, could recognize quite small doses of opiates.

2. To what extent is it permissible to compare drug effects in the test situation to conditions under which pain is actually experienced? In most methods of measuring pain threshold, there is no anxiety or fear of consequences. The subject knows that the pain will produce no permanent damage, that it is not the harbinger of medical bills or of postoperative complications, and further that it will be over shortly or can be terminated at will. Furthermore, as previously mentioned, Hill and co-workers (18, 15) have shown that anxiety about anticipated pain can be conditioned in men and animals and become so strong as to disrupt purposeful behaviour. Opiates markedly reduced this response to anticipated pain. These same workers have also shown (17) that painful electric shocks are overestimated when compared to standard shocks by anxious individuals, and that morphine eliminates this effect of anxiety. In reassured individuals such errors are not made, and morphine has no effect. Since emotional and environmental factors certainly influence the pain experience, it seems doubtful that results from experimental study can be transferred to clinical pain.

3. Is the electrodermal response an adequate index of the "response to pain"? Hardy *et al.* (12) have maintained that prevention of changes in skin resistance after narcotics was evidence of the marked action of narcotics in the "response to pain." Wikler (27) has summarized evidence which casts doubt on this view: the electrodermal response is markedly affected by barbiturates which are not analgesics; after hypnosis or frontal lobotomy, which relieve pain, various "responses to pain" may be unaffected or intensified. Wikler states (p. 443) "that neither effects on pain threshold, nor effects on measurable physiologic responses to painful stimuli are highly reliable indicators of analgesia."

As a result of these divergent results and opinions it is not possible at the present time to give any accurate statement as to the mechanism or site of action of analgesic effects of opiates or other agents such as that proposed by Hardy *et al.* (30). A useful postulate might be that analgesics interfere with the conditioned responses to anticipated pain and relieve many of the anxieties and responses associated with pain as a result of direct actions on the CNS or on conditioned responses. In any case, it seems unwarranted at this time to dissociate the pain experience into cognition of the sensation and reaction to that cognition, and attempts to do so without recognizing the possibility of their mutual interaction seem to oversimplify the experience.

This, of course, raises the question of the validity of all results based on such an attempted dissociation. Wikler (28) in an attempt to resolve conceptual difficulties stresses the operational approach in which effects of analgesic drugs have meaning only in relation to the method of measurement used. He points out that the process of measurement necessarily involves modification of that which is to be explained for purposes of control and quantification. For example, one must bring the subject into a test situation in either the laboratory or the hospital to evaluate analgesics, but the apparatus or questioning may alter the response of the subject. However, Wikler does not draw what seem the obvious conclusions, namely, that the effects of various factors in the method of evaluation should be studied, and that until such study is complete the most reliable method would be that most closely corresponding to the conditions under which analgesics will be used.

Evaluation of Analgesics using Clinical Pain

With these as well as the previous considerations in mind, Beecher and his group in Boston (3, 25) developed a method for the evaluation of analgesics under the actual conditions in which most postoperative pain occurs in patients.

They have laid down careful methods for guaranteeing that such evaluations will show clearly the pain-relieving effects of drugs under clinical conditions. Their methods involve the administration of drugs to patients and the evaluation by the patient, as well as an objective valuation of their effect in relieving pain. The following were features to guarantee correct evaluation:

(*a*) the use of postoperative patients who fulfilled the following conditions:
  (1) had sufficient trauma to warrant persistent postoperative pain;
  (2) showed no contra-indications to morphine;

(3) could give reliable information as to pain relief;
(4) were not in precarious condition, so that there was no risk from placebos, etc.

(*b*) the use of the double-blind technique in determining drug effects, by which
(1) the placebos were made similar in every possible way to the drug(s) studied;
(2) neither the patient nor the recording technician knew the agent administered;
(3) neither the patient nor technicians had the opportunity to learn to identify drugs by other effects;

(*c*) the use of the method of concurrent comparison in which
(1) agents (drugs, placebos) to be compared are given to the same patient, in so far as possible, in order to control variation between patients;
(2) agents to be compared are given in random order so that variation in pain intensity between patients and between various intervals in the same patient can be controlled.

They have used this method extensively to study the curve for the dose effect of sub-cutaneous morphine (7, 25). With a placebo, 50 per cent of patients are relieved. With 5 mgm. of morphine 61 per cent; 8 mgm. 74 per cent; 10 mgm. 74 per cent; 12 mgm. 71 per cent; 15 mgm. 83 per cent. In subsequent studies (20) they have not always found the effect of 15 mgm. (¼ grain) to be significantly better than that of 8–10 mgm. and present evidence that the higher dose causes more disagreeable side effects, at least in normal individuals. (Side effects cannot be evaluated in postoperative patients because of difficulty in differentiating them from effects of surgery and anaesthesia.) The dose effect curve of morphine requires further study, but results so far indicate that a maximal effect may be achieved at 10–15 mgm. of morphine, but that some patients will not be relieved by these doses.

They have been unable to demonstrate any greater effect from oral codeine (60 mgm.) or morphine (10 mgm.) than from a placebo (6). Large doses of acetyl salicylic acid (600 mgm.) did produce slight but significant effects. Elimination of placebo reactors from the data gave some indication that oral codeine and morphine were analgesic.

They have studied the placebo response and report that patients who consistently respond to placebo can be characterized by psychological tests, especially the Rorschach test, and by their reaction to the hospital situation. They tended to minimize their pain and when questioned thought that their care was excellent. They tended to be talkers, to be less educated and more religious. According to the authors, Rorschach tests indicated that placebo reactors were more responsive, anxious, self-centred and preoccupied with internal body processes, and more emotionally labile. They were more dependent on outside sources of stimulant than on their own mental processes, which were less mature. They had greater instinctual needs and had less developed regulation of the social ex-

pression of these instincts. They relieved their greater anxiety through their lability and outward orientation (e.g., talking).

However, it should be pointed out that there was a larger group of patients who sometimes responded to a placebo. This group also occurred in our own series of medical students. No attempt has yet been made to elucidate the nature of the factors controlling occasional placebo responses.

In line with their findings on the variation in subjective response to placebos, this group has studied the effects of morphine, heroin, amphetamine etc. on mood in normal students, in a group of clinically ill patients, and in post-addicts at Lexington. According to their findings it is only in post-addicts that opiates produce euphoria regularly. In the other groups, they most frequently caused dysphoria and unpleasant side effects. A minority of individuals experienced euphoria, but again these could be characterized by psychological testing as deviants.

Practical Implications for Anaesthesia

Beecher believes (3) that the effects he finds are primarily on reaction to pain. Actually if elevation of pain threshold in experimental studies is made equivalent with pain relief clinically, there is little disagreement as to qualities of drug effect provided only adequately controlled studies are considered. Both studies yield evidence of marked analgesia independent of suggestion only if opiates and related synthetic drugs are used. The magnitude of the effects seems greater under clinical conditions and some experimental methods do not distinguish them regularly (7). Irrespective of one's opinion as to whether it was the effects of drugs on pain sensation, pain response, or on the whole experience which were studied by Beecher's group, these results have practical implications.

Postoperative Pain

1. *The dose of morphine.* Beecher's groups have presented evidence that 8–10 mgm. of morphine provides pain relief equal or nearly equal to that of 15 mgm. (*ca* ¼ grain) of morphine, with a smaller incidence of disagreeable side effects.

Tradition has established ¼ grain of morphine for the first trial at pain relief. No doubt it is effective in most cases, but would it not be worthwhile to try to determine precisely if equal therapeutic effects can be achieved with a smaller dose? The question requires further study and cannot be answered by dogmatic statement. However, if a large number of patients are effectively relieved by 8–10 mgm. of morphine, then there are good reasons for trying it. The incidence of addiction is dependent on and directly related to the total dose injected. The incidence of side effects is similarly related to the dose injected. At the present time it would seem reasonable to try 10 mgm. of morphine initially. In most cases, it should be effective.

2. *The dose effect curve of morphine.* Beecher's group (20) has presented evidence that among postoperative patients there are some who are not relieved by 10 mgm. of morphine or any larger dose. This raises the practical question:

Is there a plateau in the dose response curve so that beyond a given dose no further therapeutic effect can be achieved and the possibility of severe depression is enhanced? Clearly this would not completely agree with the old dictum that the dose must be titrated to the patient. The character of the dose effect curve needs further elucidation in patients being treated for pathological pain.

3. *The placebo reactor.* Lasagna (22) claims there is such a thing as the small minority of consistent placebo reactors with a definite psychological make-up. If so, lacking a simple means of identifying the reactor in advance, there seems to be little possibility of practical utilization of the fact. If, however, as our data as well as theirs tend to indicate, placebo reaction is an inconsistent response of many individuals to drug therapy, then the environmental factors which tend to promote a favourable placebo reaction should be further studied. Placebo reaction might be used to support the actual therapeutic effect of drugs if conditions for its occurrence were properly understood.

In any case, the fact that placebo reactions do occur should serve as a further reminder that pharmacological effects of drugs per se are not responsible for many of the effects observed in clinical practice. Sir Thomas Lewis (23) has already pointed out that often medical practitioners tend to delude themselves in this regard. He states: "Every student [and he should include us all] should be convinced through both precept and example of the fallacy of the post ergo propter type of argument." "It is unreasonable," he continues, "that any medical graduate should be ready to believe that any improvement witnessed in a patient under their care is necessarily the result of remedies given. Such men become too easily the dupes of those who trade in proprietary remedies, or of their own too quickly gathering confidence. If, after a remedy is prescribed, one patient improves and another becomes worse, there is on this evidence no more reason to ascribe the first than the second change to the remedy, but exceedingly few qualified men know this."

What placebo reaction implies is that it is not permissible to ascribe automatically the beneficial effects of a remedy to its pharmacological prowess, since suggestion (a placebo) can often produce the same effects. The occurrence of marked effects from placebos, especially when subjective effects are sought, should serve to remind us again that, in every therapeutic effort, the response of the organism is an important, more or less independent element in any effect that may be obtained in the conscious patient. The study and rational use of this response may be of great value in the relief of pain or the treatment of disease and certainly will aid in dispelling faith in quacks and their remedies.

Pre-Anaesthetic Medication

Another point that seems to require re-examination is the basis for the use of narcotic analgesics in pre-anaesthetic medication. Lasagna (21) has shown that in normal individuals, i.e. not in pain and not post-addicts, morphine, heroin, etc. cause dysphoria rather than euphoria. What essential role, then, does morphine fill in pre-anaesthetic medication? Beecher (5), on the basis of Lasagna's work, has suggested that nothing desirable is added to pre-anaesthetic medication by morphine, and that there may be a greater risk of CO_2 accumulation and other

effects of its depressant actions. He summarizes, but has not yet reported fully, evidence that, using the double blind technique in 558 patients, experienced anaesthetists could tell when a narcotic had been given with the usual pre-anaesthetic medication only about 54 per cent of the time. Guessing should have done as well. He also states that there is little valid evidence for claims that a pre-anaesthetic narcotic reduces the dose of anaesthetic required to produce a given level of anaesthesia.

Certainly this problem requires careful direct examination by the methods permitting correct evaluation of subjective effects which have been previously mentioned. However, it does not seem scientifically correct to extrapolate from Lasagna's students to the anxious patient facing operation. Therefore, before any conclusions can be drawn, a study must be made of the effects in such patients of morphine vs. a placebo on the mood, on the ease of induction, on the dose of anaesthetic required for a given plane of anaesthesia, on respiratory exchange in the patient, etc.

The work of Hill and co-workers (18) has already clearly shown that morphine prevents the behaviour-disrupting effect of anxiety in post-addicts. If anxiety does affect any of the significant variables influencing rapidity, ease, and safeness of anaesthesia, then morphine might be expected to remove this effect. Whether the prevention of anxiety by morphine is entirely beneficial or whether it can be provided equally well by adequate sedation with a barbiturate is a question requiring study rather than speculation.

Conclusion

Some of our cherished concepts may be modified as our understanding of the mechanisms whereby drugs alter subjective phenomena grows. However, cherished concepts are less valuable than the knowledge which permits maximum use of all possible therapeutic tools. We seem to be approaching a day when the study of some drug responses in patients, though difficult, will be widely recognized as necessary, wherever it is possible, because such study provides information which cannot be obtained in any other way. Significantly it is an anaesthetist who is showing that studies in human patients do yield reliable and new information. Furthermore, if this field is like others, re-examination of concepts because of their inadequacy in practice will have value beyond the immediate range of the practical problems which initiated the reappraisal.

Résumé

On a mentionné les endroits où les drogues peuvent influencer la conduction de la douleur. A part l'effet bien connu des drogues sur les récepteurs, sur les voies de transmission, sur l'intégration à différents niveaux, on a insisté sur la possibilité des effets sur le conditionnement antérieur et l'intégration des stimuli environnants.

On a fait chez des humains des études sur la douleur expérimentale, puis devant des conclusions contradictoires et prêtant à confusion, on a repris l'étude du concept voulant que les opiacés agissent en élevant le seuil de la douleur, en diminuant les réponses à la douleur et en produisant le sommeil. On a mis en

évidence le rôle de la suggestion et de l'aptitude à identifier les analgésiques par leurs autres effets pharmacologiques causant une élévation de seuil de la douleur expérimentale. On a cité des expériences où, à l'aide de contrôles personnels, on a démontré que des analgésiques puissants n'avaient que peu ou pas d'effet sur le seuil de la douleur. On a pu démontrer, en plus, que les études expérimentales sont incapables de renseigner sur les effets possibles d'une drogue sur l'anxiété et/ou sur plusieurs réponses à une expérience clinique sur la douleur.

On a résumé le travail de Beecher, Lasagna et leurs associés de Boston; pour étudier les effets analgésiques, ces auteurs ont employé le soulagement de la douleur clinique, pathologique. On a décrit les modes de contrôle employés par ce groupe (les placebos, la technique doublement aveugle, la comparison concomittante, etc.). On a mentionné et discuté quelques-uns de leurs résultats dont les suivants, à savoir:

(*a*) que la dose optima pour la morphine est 8–10 mgm. plutôt que 15 mgm.

(*b*) que les malades non soulagés par de petites doses de morphine pourraient bien ne pas l'être par de plus fortes doses.

(*c*) que les placebos sont des analgésiques puissants mais que leur efficacité en cette matière dépend de l'état psychique du patient et de certains autres facteurs inconnus.

(*d*) que les narcotiques pourraient ne jouer aucun rôle essentiel dans la médication préanesthésique.

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PREOPERATIVE MEDICATION FOR CHILDREN*

CHARLES I. JUNKIN, M.B.**

INTRODUCTION

THE PROBLEM of premedication in children is one which vexes many anaesthetists. It is one which requires a sensible rather than a sentimental approach. It is not at all the same problem that it is in adults, although many of the answers to it can be found in any reasonable attitude towards the matter of preoperative medication in older patients.

First of all, those responsible must ask themselves what they hope to accomplish by giving drugs to the patient before operation. It is admittedly desirable to have the patient arrive in the operating room in as quiet and composed a state as possible. It is also sometimes desirable to have the preoperative medication act as a supplement to the anaesthetic agents which one plans to use. In addition, the drugs given before operation may have a beneficial controlling effect on some of the undesirable effects of the anaesthetic agent or agents.

It is in the first category that the needs and requirements of children differ from those of adults, and it is also the category over which most of the controversies on the subject rage. Children, as a rule, have not been taught, nor have they learned, either to control their fears or their emotions, or their reactions to a new and strange environment, to which they are removed from the comforting support of parents, and where they find themselves under the supervision and care of strangers. Babies react hardly at all to the break from life in the home to that of the hospital, provided their routines of feeding and rest are not altered too much. Children from two years onward, until they have started to attend school and have made a readjustment to a period of time spent away from parents and home, are the ones that usually suffer most from a trip to the hospital. Children of school age, on the other hand, usually adjust quite readily and the problem with them is in providing interest along with sympathetic care from those who attend them. No one, observing this group on the wards and in the playrooms, would suggest that they are unhappy. Indeed many of them find the hospital more interesting than the home and some have even simulated recurrences of their illnesses to get back into hospital.

The adult patient of reasonable imaginative powers usually decides to submit to operative and anaesthetic procedures of his own volition, after having the needs for operation explained and the alternatives outlined. He will probably have some fears as to the successful outcome. It is not surprising, therefore, if he is in hospital, that some form of sedation and hypnosis is provided for the evening before operation or that medication of some sort is prescribed for the period before operation, for the combined purposes of acting as a sedative and as an analgesic to obtund pain which may be disturbing to him. This premedication

*From the Department of Anaesthesia, Hospital for Sick Children, Toronto.
**Chief of the Department of Anaesthesia, Hospital for Sick Children, Toronto.

usually takes into consideration the need for employing drugs of the belladonna group, the desirable pharmacological effects of which are depression of salivary and bronchial secretions, relaxation of larynx and bronchi, and depression of vagal reflexes. The dosages of these drugs or combinations of drugs are well established for adults and are varied to fit the needs of the individual patient. The premedication might include drugs such as digitalis, which, in the cases for which it is desirable, may convert a poor operative risk into a better one. Attention to fluid and electrolyte balance might also be included in the orbit of preoperative medication but no discussion of this point will be made in this paper.

The requirements of children with regard to analgesic drugs and drugs of the belladonna group will be based on the same criteria as for adults. Analgesics may be employed for both sedation and relief of pain preoperatively. If pain is not present, drugs of the barbiturate group, usually of the short acting type, may be employed for their hypnotic effect. They will be of little value if pain is present and may even increase the degree of excitement under such circumstances.

Analgesics may also be prescribed as part of the premedication, in the absence of preoperative pain, to act as supplements to or to augment the analgesic properties of such anaesthetic agents as nitrous oxide, where the surgical procedure may result in more painful stimuli and where for one reason or another it is undesirable to employ cyclopropane or ether.

The attitude of sympathy for, and tenderness towards, the sufferings of children is almost universal. There is therefore a very natural desire to spare children many of the discomforts and disabilities that adults accept as being almost normal. The decisions with regard to operations in children are made by the parent. The child has few worries or concerns about his immediate future and unless his imagination and fears are deliberately aroused, the night before operation is no different from any other, except that he may spend it in a strange bed in a strange room. His requirements for sedation or hypnosis the night before operation are almost negligible.

One hears and reads much of the "Psychic Trauma" suffered by children, subjected to the mercy of anaesthesia for the performance of painful surgery. I must confess that I am a "doubting Thomas" as to the reality of this condition. There have been suggestions that children who have spent some time in hospital, where they have had operations under general anaesthesia, may revert to thumb sucking, bed-wetting, night fears and the like. These occurrences are chiefly observed in the pre-school group, three to five years in age. I believe they might as properly be blamed on the separation from a fond and indulgent parent, who has failed to develop a sense of stoicism towards mild discomforts and a degree of independence from constant parental attention, as on the administration of an anaesthetic. At least, in my thirty years of experience in Paediatric Anaesthesia, "trauma" has not been of sufficient moment for our child psychologists to do anything more than talk vaguely about it. Our experience certainly has not led to any scientific proof that the administration of an anaesthetic alone was the cause of subsequent psychic disorders. On circumstantial evidence it would be as

easy to prove that the condition is most prevalent in children of parents who have failed to train them for life in a society where not only they, but other children, and adults, have to be given consideration, and where occasional experiences of pain must be tolerated with some degree of stoicism.

The majority of normal children offer very little trouble, under ordinary circumstances, if the anaesthetist approaches them with intelligence and understanding. Every effort should be made to establish confidence on the part of the small patient. Where possible the procedures to be employed should be explained in terms the child can understand, and no promises of freedom from pain or discomfort should be made, unless one is sure of being able to keep such promises. More harm is done to the psyche of children by false promises and deceit on the part of adults than by a brief moment of pain, pain being one of the disciplines that few if any ever completely avoid.

A very small minority of children show unreasoning fear or an attitude of total resistance to every approach made to them. A fearful or indulgent parent is frequently the underlying cause, and in some cases I firmly believe that sedation of some sort for the parent would be a good start in approaching the child's problem. With this small group, it will make trifling difference how one approaches the initial problem of giving a sedative. Come with a syringe for intramuscular or intravenous medication, and one meets hysterical resistance which one is reluctant to overcome, no matter how gently the necessary force is applied. Rectal administration may be resented and opposed with equal resistance and may be quite ineffectual because of expulsion of the drug. Administration by mouth may be ineffective for the same reasons. All of these resistances are usually at their maximum level if a parent is present. Sometimes the removal of the parent from the scene and a decisive approach accompanied by vocal firmness often produces a surprising co-operativeness. It goes without saying that all forms of restraint or coercion should be applied as gently as possible.

Drugs, Dosage, and Methods of Administration

At the Hospital for Sick Children we do not employ a very long list of drugs for premedication. For hypnosis and sedation we use the barbiturates of the short-acting group. The choice is usually either Nembutal or Seconal. These may be given by mouth or by rectum. In some respects the rectal route is the more dependable of the two. Nembutal suppositories of ½ gr., 1 gr., 2 gr., and 3 gr. strengths are available, and are more dependable than the ordinary capsules of either Nembutal or Seconal for rectal administration. An enema the night before operation will make for more satisfactory results. This applies, also, if it is desirable to administer rectal Pentothal or Avertin as a basal narcotic agent. The former has almost supplanted the latter for this purpose, and may be given as either a 10 per cent or 5 per cent solution in water or saline in dosages of 15 to 20 mgm. per pound body weight. With this dosage, the child usually falls asleep in five to fifteen minutes following the injection. This method

of administration of Pentothal finds only limited application with us, and it is possible that we are neglecting a useful procedure because we find it more time-consuming than other methods. We cannot, as yet, bring ourselves to feel happy about leaving the injection and post-injection care to nursing personnel. If we use rectal Pentothal, we give it ourselves, and stay with the patient until the end of operation and his return to the recovery room.

At the present time, drugs of the analgesic group are used in the following order of frequency: (1) Demerol; (2) Codeine; (3) Morphine. Morphine is used only occasionally and, of the opiates, Codeine finds a wider preference. Demerol as an analgesic lends itself favourably to paediatric practice. It can be given subcutaneously, intramuscularly, or intravenously with or without atropine, the favourable actions of which it supplements. It does not seem to depress the respiratory centre to the same degree as the opiates do. It is safe to give children up to 1 mgm. per pound of body weight, but usually one-third less than this dose is used before operation. Codeine may be given by mouth, but is more effective, as an analgesic, given subcutaneously or intramuscularly. Morphine is given subcutaneously, intramuscularly, or intravenously.

As a drying agent we prefer Atropine to Hyoscine. With Hyoscine we have had, in the past, a higher incidence of excitement and undesirable cardiovascular reactions than with Atropine. Atropine in suitable dosage may be combined with Demerol or given separately, either intramuscularly or intravenously.

When these agents are given subcutaneously they should be administered at least one hour before operation. If given intramuscularly, forty-five minutes to half an hour is usually sufficient time for their effect to become evident. In emergencies, or where otherwise desirable, the intravenous route may be used, five to ten minutes before starting the anaesthetic. Much of the discomfort associated with injections may be prevented by drying the skin area after sterilization and using a sharp, small gauge needle.

In deciding what preoperative medication to order for each patient, the following factors must be taken into consideration: the physical condition of the patient, his temperament and nervous stability, the degree of apprehension or fear, the presence or absence of pain, the operative procedure anticipated, and the anaesthetic technique to be employed. The weight is more important than the age in deciding the dosage of the drugs to be used. In general, the metabolic rate rises from birth to the sixth year. It declines from the sixth year up to the onset of puberty, during which it again rises slightly, to subside to the adult level at the end of puberty. Some consideration of the age may result in an adjustment of the dose figured from weight. The physical state of the patient will also play a part in the decision. The vigorous, active, or high strung child will require the upper range of dosages, while the pale, lethargic, and listless child will require only minimal levels.

The presence or absence of pain, the anticipation of pain, or a desire to augment the analgesic properties of an anaesthetic agent such as nitrous oxide, are the only sound reasons for including analgesic drugs in the premedication. It may be desirable in many paediatric operations to provide a non-flammable

and non-explosive technique. In these cases, intravenous Pentothal and relaxants, associated with nitrous oxide–oxygen anaesthesia, with possibly trichlorethylene added, is likely to be the anaesthetic method chosen and one of the belladonna alkaloids will be a necessary part of the premedication. If the operation is to be long, larger doses of Demerol, or supplementary ones, given intravenously during operation, may prevent too much dependence on the analgesic properties of trichlorethylene and an abuse of its undeniable advantages for operations of medium or short duration. The continuous use of this agent during long operations may lead to cumulative toxic effects.

Table I giving dosages for the agents mentioned should be used with the above stipulations in mind. The doses given are well within the safe and effective range for the weights given.

Paediatric anaesthesia has made immense strides in the past decade and a half. The rapid development of intravenous agents during the war years and the

TABLE I

DOSAGE TABLE

		Hypnotics	Analgesics		
Age	Weight	Barbiturates Oral or Rectal	Opiates By Hypo or IM	Demerol IM or IV	Atropine IM or IV
1 month and under	10 lb. and under				1/400 Gr.
2 months to 1 year	10 lb. to 20 lb.	Nembutal ¼ to ½ Gr. or Seconal ⅙ to ¼ Gr.	Codeine sulph. ⅛ to ¼ Gr.	10 to 15 mgm.	1/300 Gr.
1 year to 4 years	20 lb. to 40 lb.	Nembutal ½ to ¾ Gr. or Seconal ¼ to ½ Gr.	Codeine sulph. ¼ to ½ Gr.	15 to 35 mgm.	1/200 Gr.
5 years to 12 years	40 lb. to 80 lb.	Nembutal ¾ to 1 Gr. or Seconal ½ to ¾ Gr.	Morphine sulph. 1/12 to ⅛ Gr. or Codeine sulph. ½ to ¾ Gr.	35 to 70 mgm.	1/150 Gr.
12 years and over	80 lb. and over	Nembutal 1 Gr. to 1½ Gr. or Seconal ¾ to 1 Gr.	Morphine sulph. ⅛ to ⅙ Gr. or Codeine sulph. ¾ to 1 Gr.	75 to 100 mgm.	1/100 Gr.

stimulus to civilian practice given by the younger generation of anaesthetists produced by the exigencies of war, had a profound effect, which, in the immediate post-war years, extended to a complete reorientation in paediatric methods. Endotracheal techniques, which had been a very esoteric procedure, became a commonplace; to such an extent that a new generation of anaesthetists has grown up who find it difficult to maintain satisfactory anaesthesia without the use of endotracheal methods. Relaxant drugs, of long or short action, along with intravenous barbiturates, have almost removed ether as an inhalation agent from adult practice. We still find, however, that ether is a useful and in some cases an indispensable agent in paediatric practice. For example, at the Hospital for Sick Children, Toronto, where we do anywhere from twenty to thirty tonsil operations a day, Ethyl chloride (occasionally Vinethene) Ether–oxygen anaesthesia is used in all but a small percentage of cases. Routine premedication is not used and intubation is only done on patients who are so large that control with a mouth hook might be difficult. Our nose and throat surgeons are quite happy with these methods and since postoperative pulmonary complications after tonsillectomies are non-existent in our institution, they have every reason to feel satisfied. A few children may have a short-acting barbiturate prescribed by the paediatrician before coming to hospital. Occasionally for very apprehensive children Nembutal suppositories may be given an hour or more before the time of operation, and with extreme rarity we may provide basal narcosis with rectal Pentothal. One of the chief factors which contributes to our freedom from aspiration complications is a prompt return of the cough reflex at the end of operation. No patient is removed from the operating table until he has coughed. For this reason, drugs of the opiate group are not used for our tonsil cases.

It is hard to say how much advantage is gained by the administration of barbiturates in these cases. Possibly the induction stage is quieter and more rapid than it would be without the barbiturate, but the recovery period is also somewhat prolonged. Most of these patients arrive at the Hospital as ambulatory cases, one half to one hour before the time set for operation. A few cases have been unstable enough, from their barbiturate, to have sustained falls with minor injuries. The advantages come out about even with the disadvantages, and the total effect might be just as good if the apprehensive parent were given double the dosage prescribed for the child and the drug withheld from the patient.

Cases booked for eye operations receive Atropine alone, and as many of these cases receive ether the upper ranges of dosages are usually ordered.

Newer anaesthetic techniques, which are likely to be employed in such major surgical procedures as lobectomies, pneumonectomies, the various operations presently in vogue for the correction of congenital cardiovascular conditions, and some neuro-surgical conditions, have added to the requirements for satisfactory pre-operative premedication. In most of these cases it is desirable to have the patient arrive in the operating theatre in a placid state of indifference, and for him to enjoy a prolonged period, after operation, free from the disturbance of vomiting or discomfort. Fortunately some of the newer drugs have come to our aid for satisfying these conditions. These newer anaesthetic techniques may

resort to either hypothermia or extra-corporeal circulations for the accomplishment of surgical procedures in a quiet or relatively bloodless field for appreciable periods of time. During the operation itself and for a period afterward the conditions may be precarious because of the profound disturbances in physiological function.

Dr. Code Smith (1) has proposed the following method of premedication in such cases: Cardiac patients, if in failure, are digitalized for several days. At six P.M. the night before operation chlorpromazine and promethazine hydrochloride, 1 mgm. of each for each three pounds in weight, are given by mouth to larger children and intramuscularly to smaller children. This dose is repeated at midnight and again at six A.M. Atropine in suitable doses is given intramuscularly forty-five minutes before operation. This produces a variable degree of sedation which may be augmented by doses of M(1) mixture of Laborit and Huguenard, (2) equal volumes, Chlorpromazine, Promethazine, and Demerol, yielding 8.3 mgm. Chlorpromazine, 8.3 mgm. Promethazine and 16.7 mgm. of Demerol per cc.—the average dose of which is 1 cc. per 25 pounds. This is given in fractional doses, to the point where the patient can still be aroused, but falls asleep if undisturbed. This premedication provides a basis for miminal anaesthesia with a rapid return to consciousness and an adequate voluntary respiratory exchange, with little or no postoperative vomiting, and little requirement for further postoperative sedation.

A relatively limited list of drugs for preoperative medication in children has been suggested. We believe that sympathetic and understanding care on the part of nurses and doctors with the provision of interesting and diverting surroundings do much to reduce the need for sedation in children unless pain is present. At the Hospital for Sick Children, we have found it of great advantage to have, incorporated in our operating room floor, a reception–play room for children coming from the wards, where they may spend the short time before being transferred to the operating room itself. This room has interesting murals painted on the walls and has a plentiful supply of toys for younger children and illustrated comics for the older ones. It is staffed by capable ward aids, who usually manage to gain the interest of the children and keep them diverted from a sense of strangeness or fear. A similar play room is part of our tonsil suite. Here preoperative patients show little need for sedation.

Summary

The problem of preoperative medication in children is discussed. The various conditions which influence the choice of drugs and their dosages are considered. A list of the drugs in everyday use at the Hospital for Sick Children is given. So-called "Psychic Trauma" in paediatric anaesthesia receives some consideration but is dismissed as being of negligible importance. A brief résumé of the premedication used in anaesthesia for surgical corrections of congenital heart disease is included.

RÉSUMÉ

Nous avons traité de la médication préopératoire chez les enfants. Nous avons envisagé les diverses situations qui peuvent influencer le choix des médicaments ainsi que leur dosage. Nous avons présenté la liste des médicaments en usage quotidien à l'Hôpital des Enfants malades. Nous avons porté une attention spéciale à ce qu'on appelle, en anesthésie pédiatrique, le trauma psychique mais nous avons vite discontinué cette attention parce que nous considérons que ce problème n'a qu'un intérêt négligeable. Nous avons également mentionné, en résumé, la prémédication que nous employons en vue de la chirurgie correctrice des maladies cardiaques congénitales.

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CARDIOVASCULAR DEPRESSION FOLLOWING MAJOR VAGINAL SURGERY

H. Barrie Fairley, M.B., B.S., F.F.A.R.C.S.*

This paper presents a series of 176 unselected cases of major vaginal surgery (i.e., vaginal hysterectomies, repairs, and other plastic procedures) as a basis for a discussion of the factors relating to the aetiology and logical therapy of hypotension following this type of surgery.

With the exception of four cases, all the anaesthetics have been given by members of the attending staff of the Toronto General Hospital and the surgery was performed by their gynaecological colleagues. The four cases mentioned were included since the anaesthetics were given by fully certificated anaesthetists. The work is that of seventeen different anaesthetists.

All the patients have been through the T.G.H. Private Patients' Recovery Room and, in the main, my figures are taken from the records made during their postoperative stay there. (Percentages will be quoted to the nearest unit.)

It was first established that postoperative hypotension is unusually common in this group. Defining, empirically, hypotension as a systolic pressure of 90 mm. Hg., or less, at any time in the recovery period, it was found that 101 cases (57 per cent) came under this heading. It should be mentioned that even more patients might have been added to the hypotensive group but for the fact that 23 of the cases, in the non-hypotensive group, had been given either pressor agents (11 per cent of the whole series) or blood (2 per cent of the whole series) as a prophylactic measure during the operative procedure. A recent series of 500 consecutive cases of all types of surgery, passing through the same recovery room, showed only 10 per cent to register systolic blood pressures below 90 mm.Hg. It is evident, therefore, that hypotension is unusually prevalent following major vaginal surgery—a fact already well known to many.

Causes of Hypotension

An attempt was made to consider the possible causes of this problem and to assess the extent of the contribution of each factor. These will be discussed in turn.

1. *Blood Loss*

This is not unusually gross during this type of procedure. Some surgeons reduce the volume by the injection of a solution containing adrenaline, with or without Procaine HCL. Those cases in which the blood loss seems considerable receive adequate replacement therapy as the occasion demands.

Perusal of the records in this series showed that this was unlikely to be a major factor. Of those who became hypotensive, 78 (77 per cent) had a relative bradycardia i.e., a normal or below-normal heart rate. This is not the picture which we are accustomed to see in haemorrhagic shock. Additionally, the fall in

*Department of Anaesthesia, University of Toronto and Toronto General Hospital.

blood pressure rarely occurred during the operation when blood loss would occur.

2. *Alteration in Posture*

It is reasonable to suppose that, since most of the cases in this series developed hypotension after the operation had finished, the fall in blood pressure might be due to a peripheral displacement of blood to the legs following their return from a prolonged elevation in lithotomy stirrups. This possibility merits careful examination since, if it is a major contributory factor, the treatment is simple, i.e., raising the foot of the bed after operation.

It is well known that the lower limbs are capable of holding a considerable, and variable, volume of blood. Saunders (1) has made use of this in his negative pressure method of achieving controlled hypotension and we have all used the anti-Trendelenburg position in an attempt to reduce blood loss during head and neck surgery. Bearing in mind the fact that one has initially added to the circulating blood volume (relative to the functional vascular bed) by raising the legs into the lithotomy position, at a time when the patient has just had her vasomotor tone insulted by the induction of anaesthesia, one wonders why the same volume of blood should not be returned whence it came, when the legs are lowered, and the circulatory status quo return to normal. One must consider the factors influencing venous return:

(*a*) Gravity. This is probably only of minor importance here since the legs are not lowered below horizontal and water, and therefore blood, finds its own level.

(*b*) Muscular support. Since 55 (55 per cent) of the hypotensive cases did not sustain a fall in arterial pressure until the return of consciousness (and, therefore, of muscular tone) it would seem illogical to incriminate the lack of muscular support as a major aetiological factor.

(*c*) The thoracic pump mechanism. The same argument applies here. While the patient is in lithotomy position, her respirations would be less efficient. Case and Stiles (2) have shown that the vital capacity is reduced to 82 per cent of normal in this position and one may assume that the intra-abdominal pressure is raised. Yet, the hypotension occurred when this sad state of affairs had been relieved.

(*d*) The residue of mean aortic pressure, once this has traversed the arteriolar and capillary beds. In this series, the pressure remained constant during the operation.

Of more importance are the factors influencing the size of the vascular bed in the legs. If the capacity of this is increased, then this change of posture becomes of probable significance. These factors are:

(*a*) Vascular tone. Cushny (3) has shown, by plethysmograph studies, that blood flow through the limbs is markedly increased under anaesthesia, even though the arterial pressure remains constant. Pappenheimer and Soto-Rivera (4) have demonstrated that the flow in the hind limbs is proportional to the femoral artery pressure minus the femoral vein pressure. It follows, therefore, that if the flow is increased without a rise in arterial pressure (and such a rise is

not recorded in this series) the raised flow must be related to a fall in the venous pressure and return. However, this indulgence in theory has merely shown that, when an anaesthetic is given, the peripheral vessels tend to dilate and the venous return to fall. Compensation is normally adequate and occurs as follows. The cardiac output is dependent upon the venous return. With dilated peripheral vessels, a reduced cardiac output results in a fall in arterial pressure which, in accordance with Marey's Law, supplies the necessary reflex compensating tachycardia or, simply a fall in venous return results in a rise in heart rate.

(*b*) pH changes peripherally. An increase in acidity will result in a vasodilatation. There is no adequate reason to suppose that the pH falls in the lithotomy position, in the lower limbs. The arterial pressure was maintained, in all cases in this series, while the legs were raised and removal of metabolites should have been maintained throughout. Again, a fall in pH should cause a tachycardia.

(*c*) The venular pump mechanism. This has been described by Primrose (5), who suggests that successive contractions in the minute vessels of the venous system produce a propulsive force. This is not universally accepted but, since the fall in arterial pressure seems to be related to the return of consciousness, there is no reason for assuming that this mechanism should become deficient at this particular stage.

If the fall in venous return resulting from dropping the legs to the horizontal is the main cause of our hypotension, a tachycardia should be evident. This only occurred in 11 (11 per cent) of hypotensive cases. (The fact that only 89 (88 per cent) hypotensives have been accounted for is due to the difficulty of deciding what degree of increase in rate is reasonable for a given fall in arterial pressure. A small group, therefore, did not show any significant change in rate.)

It is unlikely that venous return is deficient in many of these cases, as a primary factor. In 38 hypotensives, it was possible to observe the effect of elevating the foot of the bed, without the confusing influence of other therapy. In 30 (78 per cent) of these cases there was no improvement. Venous pressure studies might be an interesting way of confirming this point.

We return now to the other possible causes of hypotension.

3. *Prolonged Operating Time, Rough Handling of the Patient, Heat and Electrolyte Imbalance*

These factors are grouped together and it is not proposed to discuss them other than to say that they seem of little importance in this series.

4. *Carbon Dioxide Accumulation*

The respiratory embarrassment caused by the lithotomy position has been mentioned already and it is possible that this might be a contributory factor. Many of the anaesthetists used small doses of muscle relaxants and spontaneous respiration through soda-lime, as part of their technique, and one would imagine that this might cause deficient ventilation. However, these cases showed no signs of carbon dioxide accumulation during the operation. In 109 cases (62 per cent of the whole series) the patients received relaxants. Of these, 63 (58 per cent) became hypotensive after operation. In 67 cases the patients did not receive relaxants and, of these, 38 (57 per cent) became hypotensive. The use

of relaxants seems, therefore, neither to have contributed to, nor protected against, the occurrence of postoperative hypotension. Nevertheless, one feels that the use of relaxants with spontaneous respiration, in this position, requires close examination.

5. *Anoxia due to Elimination of N_2O*

Fink (6) recently showed the danger of anoxia occurring owing to the dilution of alveolar oxygen by expired N_2O, if the patient is allowed to breathe air at the end of an anaesthetic sequence utilizing this agent. He recommends that 100 per cent oxygen be given for a short period to tide the patient over this phase in his recovery. In this series, hypotension was only occasionally an immediate postoperative problem and did not respond to the routine use of oxygen therapy in the recovery room. Thus, this does not seem to be an important factor.

We are left with the two main causes of our hypotension.

6. *Pain*

This is a classical cause of hypotension and requires little elaboration. One knows that this region under discussion is well supplied with afferent fibres and that vaginal repairs rank in the forefront of painful operations. It would be reasonable to suppose that, if pain is a major contributory factor, a large group of the hypotensives would sustain a fall in arterial pressure as consciousness returns, or soon afterwards. Of the hypotensive group 55 (55 per cent) were conscious before the pressure fell. Administration of an analgesic should result in a rise in pressure. Unfortunately, this has not been the common practice, vasopressors being preferred. However, 12 (12 per cent) of the hypotensives improved following the administration of an analgesic. It is significant, though, that 7 (7 per cent) showed a fall in arterial pressure, and only entered the hypotensive group following their analgesic. Only one patient received a barbiturate–I.V. analgesic–N_2O-O_2 sequence. She maintained a normal pressure after operation.

7. *Pelvi-Cardiac Reflex*

From what has preceded, it is evident that many possible causes of hypotension exist, with this type of surgery, but there is still a significant group unaccounted for.

As mentioned earlier, 78 (77 per cent) of the hypotensives had a relative or absolute bradycardia. Systolic pressures ranged between 40 and 90 mm.Hg. and heart rates between 45–85/min. Haist states (7) that a slow heart rate, associated with a fall in arterial pressure, may be assumed to be cause and effect and probably to be a variation of the vaso-vagal theme. Certainly, the causes of hypotension, discussed earlier, are all associated with a tachycardia. Burstein (8) has described the reflex arising from the pelvic nerves' endings on the afferent side and producing an increase in vagal tone on the efferent side; it seems, in this series, that this should be considered to be the major single factor in the incidence of the postoperative hypotension. That the reflex is related to the return of consciousness, in many cases, may be only coincidence although, by that time, the vagal blocking effect of premedication and any autonomic blocking effect of the anaesthesia will have worn off.

Treatment

Assuming that the intravenous therapy has been managed correctly and that oxygen therapy, control of the airway, and elevation of the foot of the bed have been instituted, a persistent hypotension may be assumed to be due to stimuli arising from the operative site. The reflex may be broken at various levels:

1. Peripherally, on the afferent side. Long-acting local anaesthetics injected at the operative site or long-acting regional anaesthesia could possibly be of prophylactic value. The only local anaesthetic injected locally, in this series, was Procaine HCl. In all cases, this would have been eliminated by the end of the operation. It is of interest that, despite the lack of satisfactory results, 161 (92 per cent) of the whole series received barbiturate-N_2O-O_2, or a similar sequence with relaxant added. No patients received epidural anaesthesia. Sufficiently long-acting drugs, used by this route, might tide the patient over the autonomically unstable recovery period.

2. Central depression. The possible value of giving analgesics earlier and more frequently than at present has been mentioned. Variation of the general anaesthetic technique has not been adequate, in this series, to assess the relative values of the various agents. There is no reason to suppose that one agent is any better than another, given the same degree of depression and adequate ventilation. The possible blocking value of the misnamed hibernation technique might be recommended by some. Five patients received these drugs with or without barbiturate. Four came into the hypotensive group, two with obvious bradycardia.

3. Peripherally, on the efferent side. No patients, in this series, received intravenous atropine as a form of treatment. In the presence of obvious bradycardia, this is the therapeutic agent of choice, its vagal blocking effect being well established.

Inglis (9) has divided hypotension into three groups, according to whether there is fast, normal, or slow heart-rate. It appears that we are dealing with his last group, in 77 per cent of cases. Certainly, in the 46 (46 per cent) of the hypotensives who were still unconscious, atropine gr. 1/100 I.V. would have been the therapy of choice for those with relative bradycardia.

Ganglion-blocking Agents

If one assumes that the essential problem is that of a harmful reflex, which is mediated through the autonomic nervous system, and which becomes active during the recovery period, it might appear logical to use prophylactic treatment directed specifically at the autonomic ganglia. Most anaesthetists will agree that this problem is not of sufficient import to justify the use of a methonium compound or a thiophanium derivative in this connection. However, the value of intravenous procaine or lignocaine as part of the anaesthetic sequence merits consideration. Unfortunately, this series does not include any cases for which this technique was used.

The Use of Vasopressor Agents

From the practical standpoint, hypotension frequently responds to vasopressors and did so in 50 (50 per cent) of the hypotensive group. This is, essentially, symptomatic treatment and there are those who feel that this type of therapy,

not based on aetiology, is to be deprecated except in dire emergency (10). However, consideration of some of the agents used may be of value:

1. Methylamphetamine (Methedrine). This was the most popular of the pressor agents used. It has a prolonged action, stimulating both myocardium and peripheral vasculature. However, the undesirable feature of this drug is its cerebral stimulatory action, which produces postoperative restlessness. It may be used as an intravenous infusion but is less controllable, when given thus, than is nor-adrenaline. Overdosage results in prolonged hypertension.

2. L-nor-adrenaline (Levophed) (11, 12, 13). This is considered to be the physiologically active portion of adrenaline with respect to peripheral vasoconstriction. Goldenberg, Apgar, Deterling and Pines (11) were the first to publish the therapeutic effects of this agent. It is used in diluted form as an intravenous infusion, its action ceasing within a very short time after administration is stopped. This method applies the same principle but is an improvement on the I.V. adrenaline drip of Frankis Evans (14).

Churchill-Davidson and Swan (15) have compared nor-adrenaline and methedrine. It is interesting to note that they found both produce a bradycardia proportionate to the rise in blood pressure. They comment that, while it is appropriate to use the physiological component whose absence caused the hypotension, "methedrine possesses the undeniable advantage that, in most instances, it produces the desired effect without the continued attention of the physician."

3. Mephentermine (Wyamine Sulphate). For those who are impressed with the logical basis for the use of nor-adrenaline but who require a longer action, mephentermine has many advantages. Its peripheral action and the duration of its effect, comparable with that of methedrine, without the excitatory action of the latter, render it a valuable agent.

In closing the subject of vasopressors, one might mention Inglis's recommendation that they be used for his middle group of hypotensives with normal heart-rates.

Hypotension with Tachycardia

Mention should be made of the 11 (11 per cent) cases in the hypotensive group who showed definite tachycardia. While some of these may have been suffering from one or more of the common causes of "shock," mentioned above, there is always the odd case which does not respond to routine therapy. The possible value of Ouabaine, or Strophanthin G, is mentioned in passing here. Horton and Armstrong Davidson (16) have renewed interest in the use of this cardiac stimulant in shock and suggest, among other indications, its use to restore blood pressure with or without blood volume replacement, "especially where there is peripheral vaso-constriction or tachycardia and when vasopressor drugs might then be contraindicated." It is felt that the number of occasions on which this agent would be of real value is small but that it should be part of the resuscitation armamentarium. Doses of 0.5 mgm. are recommended.

Influence of Age on Incidence of Hypotension

This influence is difficult to assess in the present series and consequently mention has not been made of it before. Twenty cases (11 per cent) from the whole series were below 40 years of age and might be assumed not to have

developed peripheral vascular disease. Of these, 65 per cent (i.e., 13 out of 20) became hypotensive. In the larger, over 40, group any or all of whom may have had peripheral vascular disease, 56 per cent (i.e., 88 out of 156) became hypotensive. It is doubtful whether one may draw any conclusion from this.

Minor Vaginal Surgery

Comment here is with special reference to dilatation and curettage. Although the very large number of patients who have undergone this procedure and passed through the recovery room, have not been included in the present series, it is of interest that many have been noted to show hypotension with a relative bradycardia after operation. There being no element of pain present, it is considered that these exemplify the pelvi-cardiac reflex mentioned above. This conclusion has been supported by their response to intravenous atropine.

Summary

A series of 176 cases of major vaginal surgery have been followed through their immediate postoperative course and the incidence of hypotension in association with a relative bradycardia has been found to be high. The possible aetiology and logical therapy have been discussed.

Acknowledgments

I am indebted to Dr. J. McArthur for drawing my attention to the high incidence of hypotension following this type of surgery, to Dr. R. E. Haist for his opinion on the physiology of the postural changes involved, and to the recovery room nurses of the Toronto General Hospital Private Pavilion, whose very adequate records were of the maximal assistance.

Résumé

On a surveillé les suites post-opératoires immédiates de 176 cas de chirurgie vaginale majeure; on a trouvé une incidence élevée d'hypotension associée à une bradycardie relative.

On a essayé de trouver les causes possibles de ces symptômes; les causes adjuvantes pourraient être: une perte de sang, un changement de position, un temps opératoire prolongé, des manipulations brusques, un déséquilibre de la chaleur et des électrolytes, une accumulation de CO_2, une anoxie due à l'élimination de N_2O et enfin la douleur.

Ces causes étudiées, il reste quand même un groupe imposant que l'on ne peut expliquer. Les causes d'hypotension, discutées plus haut, sont toutes associées à une tachycardie. On a supposé que ces patients souffraient d'une dépression cardiovasculaire à cause d'un réflexe pelvicardiaque perçu par les terminaisons nerveuses pelviennes sur le côté afférent et produisant, sur le côté efférent, une excitation vagale telle que décrite par Burstein (8); ces stimuli naissent au site de l'opération.

Le traitement logique de ces conditions serait de bloquer ce réflexe à différents niveaux:

(1) à la périphérie, sur le côté afférent, par une injection d'un agent anes-

thésique local à longue action au site même de l'opération ou encore par une anesthésie régionale à longue action.

(2) par une dépression centrale en donnant des agents analgésiques plus tôt et plus fréquemment.

(3) à la périphérie, sur le côté efférent, en donnant de l'atropine par voie intra-veineuse.

L'emploi d'agents ganglioplégiques tels que la procaïne ou la lignocaïne comme adjuvants d'une anesthésie qui a pour but spécifique de bloquer le système nerveux autonome mérite d'être pris en considération.

L'emploi de vasopresseurs est un traitement purement symptomatique mais, au point de vue pratique, il s'est avéré efficace en corrigeant l'hypotension dans 50 pour cent des cas de ce groupe de chutes de pression.

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PACATAL* IN ANAESTHESIA: A PRELIMINARY REPORT**

JOHN I. DAVIES, M.D., F.R.C.P.(C), F.F.A.R.C.S.
DONALDA H. M. HUGGINS, M.D.
CHRISTOPHER F. WOLKENSTEIN, M.D.***

THE INTRODUCTION of the lytic cocktail into clinical use by Laborit and Huguenard in 1951 (1), and the excellent results claimed, stimulated interest in so-called "ganglio-plegic" drugs and "l'hibernation artificielle." Whatever one's opinion may be regarding the usefulness and rationale of such techniques, it must be agreed that new, powerful, and interesting drugs have been added to the anaesthetist's armamentarium. One of these, chlorpromazine, has been the cause of lively controversy, mainly because some investigators seized on the unusual properties of the drug to sustain hypotheses of their own making (2). That at least 2,000 papers have been published on chlorpromazine proves the widespread interest in this drug. In Canada, these include valuable papers by many investigators.

The concept of tranquillizing action, apart from any effects of hypnosis or analgesia, has emerged. Recently the term "Ataraxia" meaning "calmness, untroubled by mental or emotional excitement" has been used by Fabing (3, 4). Many well-established drugs such as the narcotics and barbiturates may produce this effect in addition to their other well-known properties.

Because of the stress and strain of present-day living, drugs having a predominately tranquillizing effect have extensive clinical application. The narcotics, barbiturates, and hypnotics because of undesirable effects have limited therapeutic usefulness when only a tranquillizing effect is desired. Drugs such as Serpasil (5, 6), Miltown or Equanil (Meprobamate) (7, 8) and more recently Suavitil (Benactyzine HCC) (9, 10), in spite of some disadvantages, have been found very useful clinically in producing relief of nervous tension. As a result of these successes, drugs of a similar nature can be expected to flood the market. Because the tranquillizing properties of these drugs may be used to advantage by our specialty, anaesthetists should maintain an interest in the investigation of such drugs in the hope that eventually an ideal one may be found.

The phenothiazine derivatives deserve special consideration as members such as chlorpromazine, promethazine, Diparcol, and Avomine have already found a place in medicine.

In a search for less toxic phenothiazine derivatives, Schuler and Nezel synthesized eight compounds each having a terminal piperidine ring attached to the nitrogen atom of phenothiazine. These compounds were investigated by

*A phenothiazine derivative.

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***From the Departments of Anaesthesia, University of Manitoba, Winnipeg General Hospital, and the Childrens' Hospital, Winnipeg.

Nieschulz in 1954 and the most promising one, P 391 or Pacatal, was subjected to an exhaustive and detailed study (11).

Pacatal is N-methylpiperidyl-3-methylphenothiazine and its structural formula is shown in Figure 1.

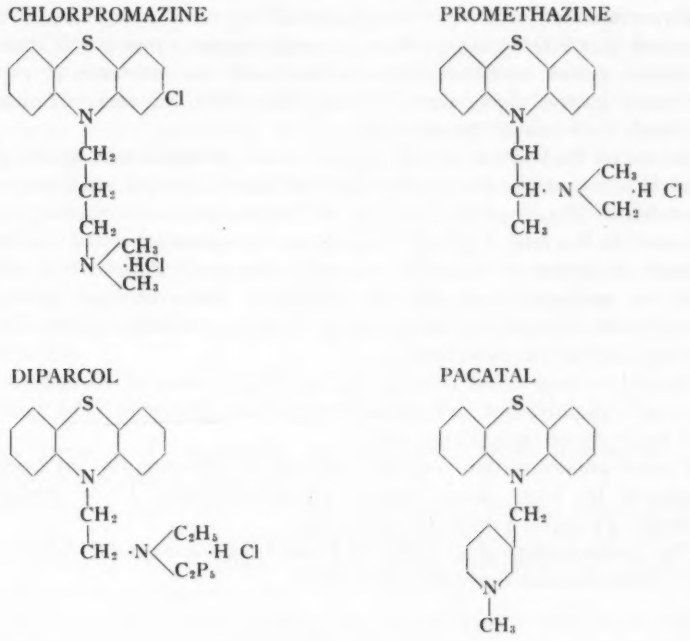

FIGURE 1. Structural formulae of some phenothiazine derivatives.

PHARMACOLOGY

Pacatal has been extensively investigated in various laboratory animals by many workers including Nieschulz (12), Gadermann (13), Donat (14), and Horatz (15) and their findings are briefly summarized.

Pacatal was absorbed and eliminated quickly.

LD_{50} varied from 20 mg./kg. i.v. in rabbits to 750 mg./kg. s.c. in mice. Daily administration of up to 20 mg./kg. orally to dogs for six months produced no untoward effects.

Circulatory effects included decreased capillary permeability, vasodilatation, and vasodepression. In anaesthetized dogs, doses of 5 mg./kg. produced moderate depression of blood pressure (20 mg.Hg) which could be reversed by vasopressor agents, but was accentuated by Pendiomid or Arfonad. The main effects on the heart were "intravascular anaesthesia," protection against epinephrine-induced and hypothermic arrhythmias, and coronary vessel dilatation in the isolated guinea-pig heart.

Pacatal seemed to depress the sympathetic nervous system producing partial adrenergic blockade and some measure of adrenolytic activity, in that the pressor, but not the local vasoconstrictor or hyperglycaemic effects of adrenalin was inhibited. Blood sugar levels were not usually affected. Nor-epinephrine was not blocked. The carotid sinus reflex was inhibited.

Parasympathetic activity was apparently depressed as salivary secretions were lessened, gut activity was reduced, vasodepression from vagal stimulation was inhibited, gastric secretions were reduced, and the incidence of experimentally produced gastric ulcers was lessened. The effects of acetyl choline were unchanged. Mydriatic action was slight.

Action on the central nervous system was a predominantly quieting effect and marked sedation was seen only following higher doses. Pacatal was a mild anticonvulsant. The hypnotic activity of barbiturates was prolonged without a decrease in the rate of fall of the plasma concentration of the barbiturates. The anaesthetic action of ether was markedly increased. Pacatal is a mild analgesic and the analgesic properties of morphine, Demerol, and phenacetin were potentiated. Pacatal was an effective local anaesthetic agent. The action of succinylcholine was potentiated.

Pacatal in large doses (50 mg./kg. in rabbits) lowered normal body temperature only slightly, but had strong antipyrexial properties. The basal metabolic rate was reduced up to 23 per cent.

Pacatal administration was not followed by albuminuria but haematuria was produced by large doses (as by chlorpromazine). Liver damage was not reported. Pacatal is a mild anti-histamine.

The pharmacological activities of Pacatal are summarized and compared with some phenothiazine derivatives in Table I.

TABLE I

SUMMARY OF PHARMACOLOGICAL ACTIVITY OF SOME PHENOTHIAZINE DERIVATIVES
(after Horatz, 17)

	Phenergan	Diparcol	Largactil	Pacatal
Antihistamine	+++	+	+	++
Parasympathetico-lytic	++	+	+	++
Sympathetico-lytic	+	+++	+++	++
Blood pressure lowering	+	+	++	++
Blood pressure stabilizing	++	+	++	++
Analgesic	+	+	++	++
Metabolism lowering	+	++	++	++
Temperature lowering	+	++	+++	+
Local anaesthetic	+++	+	+++	+++
Anticonvulsant	++	+++	+++	+

Strong action: +++ Medium action: ++ Weak action: +

CLINICAL STUDIES BY OTHER INVESTIGATORS

It must be remembered that accurate inferences cannot always be made from the results of experiments with animals (16). Apart from species differences,

much higher doses may be given to animals with apparently different results. Also animals may be under barbiturate anaesthesia to facilitate some recording, such as blood pressure changes.

Horatz first reported the clinical use of Pacatal in 439 cases (17) and later in 1,144 patients undergoing surgery (18). Premedication was with repeated 50 mg. doses of Pacatal, morphine being withheld except in the case of severe pain. The total dose and route of administration depended on the severity of the procedure but his dose schedule included barbiturates, Dolantin and in children atropine. This series included 403 pulmonary, 21 cranial, and 20 cardiac operations. Minor cases were "pretreated" and in major cases Pacatal was used for "potentiation of narcosis." Horatz later enlarged his series to over 5,000 cases (19). He combined Pacatal with Pendiomid for controlled hypotension, and with refrigeration in place of the "lytic cocktail," claiming that using a single drug is advantageous. He used Pacatal for many hours preoperatively. Good results were claimed but hypersecretion after thoracic surgery, hyperpyrexia (easily controlled by amidopyrine), and increased difficulty in judging the patient's postoperative condition were occasional disadvantages. Toxic potentiation of narcotics was noted.

Other Reported Uses

Pacatal has been used successfully in combination with barbiturates in "Schlaftherapie" or therapeutic sleep (20).

In psychiatry encouraging results were obtained with 50 patients suffering from mild anxiety states (21) but hypochondriacal schizophrenics were not affected (22). Manics showed a wide variation in response and best results were claimed in anxiety states and drug addiction (23).

Lindsay (24) has used Pacatal in more than 50 psychiatric patients in office practice and noted its good sedative action. The treatment was later modified because of occasional complaints of "blurred vision," found to be caused by difficulty in accommodation.

Good results have been reported by Kline (25), but complications including hyperpyrexia, dryness of the mouth, urinary retention, and lack of therapeutic benefit were reported.

Pacatal has been used to potentiate the analgesic action of morphine in cases of inoperable carcinoma (26).

The drug has aroused wide interest mainly in Germany. At least thirty papers have been published and more can be expected in the near future.

Clinical Study

Pacatal is at present available as tablets containing 50 mg. or 25 mg. of Pacatal base (as Hydrochloride), and as ampules (2 cc.) containing 25 mg. Pacatal base (as Acetate) per cc. The pH of the solution is 5.6 to 5.8 at 25°C. The solution is not physically compatible with solutions of Pentothal, Demerol, chlorpromazine, promethazine, muscle relaxants, or with normal saline, but can be diluted with distilled water or 5 per cent Dextrose water.

Local Effects

Pacatal, like most phenothiazine compounds, is irritating to tissues. Early in the series, Pacatal was given intravenously undiluted to 35 patients and a transient phlebo-thrombosis occurred in two patients. Since we began diluting Pacatal solution with at least three times its volume, no phlebo-thrombosis has resulted from its use. Following intramuscular injection in 106 patients, 84 complained of sore buttocks after operation. This was particularly noticeable in children when relatively large doses had been given. Because of this, oral administration is preferred for premedication, particularly since this route gives equally satisfactory results. Oral administration produced no nausea, vomiting or gastric discomfort.

Dosage

Early in the series, Pacatal was given intravenously to adult patients in doses ranging from 25 to 250 mg. It was found that the small doses produced no obvious effects, and that the large doses used produced no more of the desirable effects than moderate doses.

The usual dosage evolved for adults was 100 to 200 mg. (about 1 mg. per pound of body weight) and relatively larger doses for children (about 1½ mg. per pound of body weight) by whatever route was chosen.

In our present series, Pacatal was given to 352 patients ranging in age from 2 to 86 years. There were 204 children and many of the adults were "poor risk" patients. The drug was used in the following ways:

Present Investigation

1. H.S. Sedation—56 cases.
2. Premedication either alone or in various combinations with Atropine and Demerol—204 cases.
3. Following usual premedication, in conjunction with anaesthesia, either immediately preceding induction or during the course of anaesthesia—148 cases.
4. Postoperatively—42 cases.

Results

Of the 56 patients who had received Pacatal orally at bedtime, 47 slept well.

Patients who had received the usual dose of Pacatal about 1 to 1½ hours previously usually arrived in the operating room in a tranquil frame of mind. A few were asleep when left undisturbed, but awoke readily. Most rested peacefully and though alert mentally, they appeared to be unusually calm in the acceptance of their surroundings. This was particularly noticeable in the case of children who would tolerate venipuncture for Pentothal administration with unusual equanimity.

Sedation was considered excellent in 26, good in 142, and poor in 37.

In most cases salivary secretions were greatly reduced, particularly in adults, and although the mouth was not completely dry, excessive secretions were not troublesome during subsequent anaesthesia. Secretions, when present, were of

normal consistency and not tenacious. In children, particularly during ether anaesthesia, secretions were sometimes troublesome. Consequently in addition to Pacatal, atropine was given in half the usual dosage and this combination was satisfactory in most cases.

In 53 cases, Demerol (in dosage ½–¾ mg. per pound) was given as well as Pacatal. Sedation was better than would be expected from the use of either drug alone, but no potentiation of the toxic effects of Demerol was seen, suggesting that the increased sedative effect was one of "combination" rather than potentiation. No respiratory or cardiovascular depression was seen, even in elderly subjects.

Whenever the drug was given immediately before operation, alone or after the usual premedication with atropine and Demerol or morphine, the patients within three minutes become more sedated without increased respiratory depression. Even intravenous doses up to 250 mgm. given rapidly produced little effect on blood pressure or pulse rate.

No peripheral anti-sympathetic effects were seen. The size of the pupil was not affected; there was no congestion of nasal mucous membrane; no venous dilatation; no warm dry flushed skin; and sweating, if present when Pacatal was given, was not abolished.

No amnesic effects were attributed to the drug. Hyperexcitability did not occur.

Effects during Anaesthesia

Central Nervous System Effects

The induction dose of Pentothal to the loss of the lid reflex was usually reduced by about 20 to 30 per cent of anticipated dose, and this estimated reduction of Pentothal dosage was confirmed in 9 patients who had received Pentothal for induction a short time previously.

All inhalation anaesthetic agents commonly used were slightly potentiated in that the course of anaesthesia was unusually smooth and patients could be maintained in light anaesthesia with minimal amounts of agents. So-called "automatic" anaesthesia was usual. If reflex activity returned during too light a level of maintenance, the patients' reactions were slight, and not a vigorous protest. Return to an adequate level of anaesthesia was easily accomplished. This was particularly noticeable during tonsil dissections. There was no increased respiratory depression and it was felt (as in the case of narcotics) that Pacatal increased the duration of action of agents rather than their depression of vital functions. When Pacatal was given during anaesthesia, similar results were noticed and the level of anaesthesia apparently deepened. The action of muscle relaxants did not appear to be affected.

Cardiovascular Effects

During anaesthesia changes in blood pressure or pulse rate were seldom seen and could usually be attributed to definite operative or anaesthetic causes. However, the hypotensive effect of Pentothal was sometimes exaggerated particularly

in elderly hypertensive subjects. In 9 such cases, a normal response to small doses of Methedrine was obtained. Protection against untoward reflex activity was hard to evaluate as Pacatal was not used to cover errors in anaesthetic technique and any excessive blood loss was replaced.

Postoperative Period

Increased postoperative depression and delay in regaining consciousness was not seen except when patients had been given usual (but, following Pacatal, relatively large) amounts of Pentothal or ether. This depression was not seen when further experience with the drug showed that only minimal amounts of anaesthetic agents were necessary to maintain adequate depth of anaesthesia.

Though conscious in response to questioning or painful stimuli, most patients usually slept peacefully if left undisturbed. This tranquil state lasted for about 4½ to 5 hours after administration and narcotics were rarely needed. When this tranquil state wore off, most patients rested quietly for about another six hours, particularly if usual doses of analgesics had been given to combat pain.

Postoperative restlessness was unusual, particularly in children following minor operative procedures.

The incidence of immediate postoperative emesis appeared to be greatly reduced; it occurred in 10.5 per cent of cases. Ten patients with a history of post-anaesthetic hyperemesis were given Pacatal during anaesthesia and three did not vomit after operation.

No profound hypotension was seen, but a slight hypotension of 10–15 per cent of expected blood pressure levels was sometimes seen, when moderate amounts of Pentothal or ether had been given during anaesthesia.

In the postoperative period, Pacatal was repeated 27 times when its main effect had apparently worn off. Emesis was controlled in 16 out of 18 patients and restlessness was improved in 6 out of 9 cases.

There was no increased incidence of urinary retention after operation and no case of albuminuria or haematuria attributable to the use of Pacatal. Increased secretions of the respiratory tract or hyperpyrexia were not seen. There was no clinical evidence of liver damage or alteration in the blood picture.

Other Uses

In Obstetrics

Pacatal was given in doses of 150 mg. intravenously to 15 multigravida during the second stage of labour. No analgesic effects were seen and no potentiation of Demerol which had been given to 12 patients. Course of labour was very rapid (possibly even accelerated) and all delivered normally. No effects on the babies were seen. The patients were tranquil after delivery.

Pacatal 100 mg. was given intravenously before spinal anaesthesia to five patients. Hypotension in 3 cases responded rapidly to Methedrine. A fair sedative effect was obtained in all cases, but this seemed to be abolished when Methedrine was given.

Pacatal was given 50 mg. orally t.i.d. to 12 patients suffering from emesis gravidarum. Seven were cured, 3 helped and 2 not affected.

Status Epilepticus

Pacatal (100–200 mg. 1M) was given to two patients in Status Epilepticus, having very frequent seizures uncontrolled by large doses of Dolantin, paraldehyde, and barbiturates. In both cases the number and severity of the attacks were greatly reduced but only a slight increase in somnolence occurred.

Shock

Although Pacatal is not a powerful vasodilator or anti-adrenergic agent, the work of Martin (26) prompted our use of Pacatal in one case of profound shock caused by severe head injury. The role of Pacatal combined with hypothermia was difficult to evaluate but the patient made a good immediate recovery.

E.E.G. and E.K.G. Investigation

Pacatal was given intravenously to a healthy 176 lb. (75 Kg.) adult male and the E.E.G. and E.K.G. findings were as follows.

The resting E.E.G. showed normal alpha activity at 11 c/s.

Drug A (Pacatal 100 mg.) showed no noteworthy effect.

Approximately three minutes after the administration of Drug B (a second dose of Pacatal 100 mg.) the alpha activity was replaced by slow activity at 5 c/s and 7 c/s.

The pattern was *not* that usually associated with drowsiness or sleep; it does not resemble the effects of barbiturates and is worthy of further investigation.

The E.K.G. tracings showed no abnormalities.

Discussion

The precepts stated by Melville (27) and by Gilbert and co-authors (28) provided valuable guidance in attempting to evaluate Pacatal.

Our observations on the effects of Pacatal have been varied and difficult to assess. The main reason for this was that, compared with the usual definite effects of chlorpromazine, those of Pacatal were less obvious—almost subtle or unobtrusive. At first, we considered that Pacatal produced very little effect, but as the series enlarged we became more convinced of the usefulness of Pacatal.

In this clinical study it was inevitable that comparison would be made with the effects of chlorpromazine. Although the actions of both drugs are similar in many respects, Pacatal appears to have a potency roughly 1/10 to 1/15 that of chlorpromazine. Pacatal has a much milder action and more even effect.

In comparative doses Pacatal produces less drowsiness and detachment from surroundings, but at least equal tranquillizing effects. Fewer side effects were seen with Pacatal as hypotension was unusual and never profound. The peripheral sympatheticolytic effects of chlorpromazine were never seen. Pallor has been observed in ambulatory patients and in anaesthetized patients when blood loss was excessive. In such cases absence of hypotension and tachycardia suggested that the peripheral vasoconstriction was adequate in maintaining normal cardiac output. If the carotid sinus reflex was depressed it was not apparently abolished.

It is impossible without determinations of blood levels to determine whether in fact potentiation of narcotics and anaesthetic agents does occur, or whether the augmented effects were caused by "combinations." This drug lacks specific action as no one pharmacological effect is outstanding. Although the action of Pacatal appears to be mainly central, it would be invaluable if further pharmacological studies could determine its site of action.

In the postoperative period, decreased restlessness, nausea, and vomiting were a definite advantage. Compared with chlorpromazine, postoperative depression was seen less regularly and when present was of less profound and shorter duration. The further 5 or 6 hours of restfulness were of benefit to patients.

Pacatal may have specific usefulness in "poor risk" patients, as its use may fulfil the concept of almost homeopathic doses in the administration of other agents (29), with minimal side effects.

It is possible that further investigation will confirm its pharmacological value in preventing arrhythmias during hypothermia and intracardiac operations.

In view of the low toxicity of Pacatal and its many desirable features we feel that this agent merits further and more extensive investigation to determine its place of usefulness in anaesthesia and related fields.

Summary

The history of the introduction of a new phenothiazine derivative into medicine has been reviewed and allusion made to the concept of ataraxic drugs.

The pharmacology of Pacatal in animals has been briefly reviewed and also the clinical findings of other investigators.

This report deals with observations made on the use of Pacatal for 352 patients undergoing surgery, 44 obstetrical patients, and other uses in 3 patients.

Doses of 100–200 mg. (1 mg./lb.) were found effective irrespective of the route administered.

Favourable effects noted include: good preoperative sedation, tranquillization more noticeable than hypnosis, ease of control of anaesthetic procedure (between anaesthesia and analgesia), possibility of decreased amounts of anaesthetic agents, antisecretory effects enabling the use of atropine to be avoided in adults, normal response to vasopressors, tranquil postoperative course, decreased incidence of postoperative nausea and vomiting.

The drug was relatively non-toxic to the cardiovascular and respiratory systems. Dangerous potentiation of narcotics was not seen.

Irritation at the site of injection can occur. The drug is rapidly eliminated and postoperative depression is unusual. However, if anaesthetic agents (particularly Pentothal or ether) are used in moderate amounts following Pacatal administration, postoperative depression may be prolonged for two to three hours.

Further investigation of this agent is considered desirable.

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Résumé

Tout en repassant l'histoire de l'introduction d'un nouveau dérivé phenothiazine, on a fait allusion au concept de médicaments "ataraxiques."

On a également revu rapidement la pharmacologie de "Pacatal" chez les animaux ainsi que les résultats des travaux cliniques réalisés par d'autres chercheurs.

Ce travail se base sur des observations faites avec "Pacatal" en chirurgie chez 352 patients, en obstétrique chez 44 patients et en d'autres circonstances chez 3 patients.

Sans tenir compte du mode d'administration, des doses de 100 à 200 mgms (1 mgm/lb) ont été jugées efficaces.

Parmi les effects favorables, on note: une bonne sédation pré-opératoire, un calme plus marqué que l'hypnose, un contrôle facile de l'induction anesthésique (entre anesthésie et analgésie), un besoin moindre d'agents anesthésiques, des effets antisécrétoires allant jusqu'à éviter l'emploi d'atropine chez l'adulte, une réponse normale aux vasopresseurs, une période post-opératoire tranquille, des nausées et des vomissements post-opératoires diminués.

On a trouvé que le médicament ne produisait relativement pas de dépression sur les systèmes respiratoires et cardio-vasculaires; il n'a pas produit non plus de potentialisation dangereuse des narcotiques.

A l'endroit de l'injection, une irritation peut apparaître. Le médicament est rapidement éliminé et il est rare d'observer une dépression postopératoire. Cependant, si certains agents anesthésiques (particulièrement le pentothal ou l'éther) sont employés en quantité modérée après une administration de Pacatal, la dépression post-opératoire peut être prolongée de 2 à 3 heures.

De plus amples recherches sur cet agent seraient désirables.

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LA CHIRURGIE ET L'ANESTHESIE CHEZ LES VIEILLARDS*

LÉON LONGTIN, M.D., F.R.C.P.(C)**

Qu'est-ce que la vieillesse?

La vieillesse n'est pas une maladie, mais un état d'affaiblissement des forces et des facultés survenant ordinairement dans un âge avancé.

La sénescence est une évolution normale qui, d'après A. Bourguignon (8), s'accompagne d'une perturbation des protéines, d'une hypercholestérolémie et d'une deshydratation progressive des colloides tissulaires. Dans le vieillissement, les hormones hypophysaires perdent leur énergie chimique et deviennent irréversibles; les réactions intercellulaires également prennent progressivement un caractère d'irréversibilité.

Le nombre de vieillards augmente-t-il?

Lors de sa cinquième session ordinaire (Vienne, 5–8 septembre 1955), le Comité régional de l'OMS a aussi discuté la question du vieillissement des populations. Nécessitant des modifications des services de santé publique, ce problème a donné lieu à un échange de vues animé. Au cours de cette discussion non-officielle, certains chiffres éloquents ont été fournis: en Suède, pays qui compte près de 7 millions d'habitants, 10 pour cent environ de la population ont dépassé l'âge officiel de la retraite, qui est de 67 ans, et cette proportion va en augmentant. En Grande-Bretagne, la plupart des malades que les médecins praticiens visitent à domicile sont âgés de plus de 65 ans: pour une visite à un malade de moins de 65 ans, on compte plus de 3 visites à des malades plus âgés. Pour les infirmières visiteuses, la différence est encore plus grande: pour une visite à un malade de moins de 65 ans, on compte 8 visites à des malades plus âgés.

Depuis le début du siècle, d'après Sidney E. Jiffren (4), la population a doublé aux Etats-Unis. Durant le même temps, le nombre de personnes de 65 ans et plus a quadruplé et le nombre des personnes âgées de 80 ans et plus aurait atteint le chiffre de 1,670,000. Au dire de cet auteur, les Etats-Unis d'Amérique compteront, en 1960, 5,570,000 personnes âgées de 65 ans et plus; ce chiffre représentera 13.8 pour cent de la population; de ce nombre, 2,175,000 seront âgées de 80 ans et plus.

Donald T. Bosch (9) a abordé le problème différemment et vient à affirmer que, de 1930 à 1940, la population des Etats-Unis a augmenté de 7 pour cent; chez les personnes âgées de 65 à 75 ans, l'augmentation a été de 35 pour cent et, chez les personnes âgées de 75 ans et plus, l'augmentation a été de 38 pour cent. Ces calculs permettent d'affirmer que, en 1980, 20 pour cent de la population des Etats-Unis sera âgée de 60 ans et plus, soit 40,000,000 de vieillards sur une population de 200,000,000.

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**Professeur Agrégé d'Anesthésiologie de l'Université de Montréal.

En Angleterre, le Professeur Mackintosh (7) a comparé les pourcentages de vieillards, par rapport à la population, existant à diverses époques dans son pays, il donne les résultats suivants: comparé à ce qu'il était en 1891, le pourcentage des vieillards aujourd'hui a exactement doublé; puis, il prévoit que, en 1980, en Angleterre tout comme aux Etats-Unis, 20 pour cent de la population sera âgée de 65 ans et plus. Au Canada, il y a 100 ans, la longévité était de 40 ans; aujourd'hui, elle est de 66.3 chez les hommes et de 70.8 chez les femmes; il y a tout lieu de croire que la longévité suivra la même courbe; en conséquence, en qualité d'anesthésiste, il nous faut nous attendre et nous préparer à recevoir sur la table d'opération un nombre de vieillards toujours croissant, au cours des années à venir. Dans le passé, les vieillards soumis à la chirurgie étaient peu nombreux et étaient considérés des risques sérieux; depuis quelques années, le nombre de ces candidats à la chirurgie a augmenté et le risque semble moins grand. Demain, selon les statistiques, le nombre de ces candidats sera encore plus grand; sera-t-il possible pour les anesthésistes, de diminuer encore le risque opératoire chez les vieillards, de diminuer la morbidité et la mortalité en dépit de l'audace grandissante de la chirurgie sur le nombre et l'étendue des opérations.

A quoi tient le succès de la chirurgie chez les vieillards?

Peut-être encore plus que dans le reste de la chirurgie, le succès de la chirurgie chez le vieillard tient au succès de l'anesthésie; ce dernier succès tient à l'évaluation clinique exacte du malade, à sa préparation à l'acte opératoire, à la minutie des soins per- et post-opératoires. En général, l'étude clinique de ces malades peut-elle renseigner sur les chances de succès d'une opération, peut-elle permettre de proscrire ou de limiter la chirurgie, permet-elle de dépister les principales déficiences à combler?

L'âge de 70 ans et plus influence-t-il beaucoup la mortalité? La présence de pathologie cardiaque chez le vieillard diminue-t-elle les chances de survie en cas de chirurgie? Les pulmonaires chroniques constituent-ils un risque plus élevé pour la chirurgie? Est-ce que l'état cardio-pulmonaire permet d'établir un pronostic de succès? Enfin, quelles sont les complications les plus fréquentes et les plus graves? Quelles sont, par ordre de fréquence, les causes de mort?

Les auteurs s'accordent pour répondre à ces questions et James Mithoefer (3) résume ainsi les réponses: La cause la plus fréquente de la mort est la défaillance cardiaque, la deuxième est l'atélectasie pulmonaire et la pneumonie. En ce qui concerne la fréquence des complications, la première est la complication pulmonaire: 38 pour cent des cas, la deuxième: les complications cardio-vasculaires, 25 pour cent, la thrombose, 5.4 pour cent.

Après 70 ans, l'âge n'influence plus le pronostic, les vivants seraient aussi artério-scléreux que les morts. Au dire du même auteur, on pourrait affirmer que l'angine de poitrine assombrit le pronostic de la chirurgie chez le vieillard et que les pulmonaires chroniques paient un tribut plus élevé à l'atélectasie pulmonaire et à la pneumonie.

Quelles sont les plus fréquentes déficiences que la clinique permet de déceler chez les vieillards?

D'après Harry Gross (18), la première est la déshydration et la seconde, la

malnutrition pouvant aller jusqu'à la cachexie. La malnutrition peut se rencontrer plus souvent chez les gens moins fortunés; ils sont habituellement d'un poids inférieur à la moyenne et dans un état chronique de malnutrition.

La diète de ces malades est insuffisante à plusieurs points de vue. Parfois, c'est leur état financier qui ne leur permet pas d'acheter une alimentation assez abondante et surtout assez variée en viandes et en fruits. Parfois, ils vivent seuls, ils sont obligés de préparer leurs repas et, souvent, ils n'ont pas les facilités de cuisson et d'emmagasinage convenables, alors ils se contentent d'aliments préparées d'avance ou du moins faciles à préparer: aliments composés surtout d'hydrates de carbone. Ou encore ils sont porteurs d'une dentition insuffisante ou d'une dentition difficile et ces gens s'abstiennent de manger de la viande et choisissent instinctivement des aliments à mastication facile: des hydrates de carbone. Les vieillards ont souvent le dégoût du lait; ils sont ainsi privés d'une source précieuse de protéines et de calcium.

En plus, certains états dépressifs et l'anxiété peuvent conduire assez souvent à l'anorexie et entrainer une malnutrition sérieuse. La solitude, l'insécurité, le sentiment d'une dépendance impossible à éviter sont des facteurs émotionnels qui conduisent parfois à des gastrites et cette gastrite ennuyeuse, douloureuse, force les porteurs à limiter leur alimentation. D'autres états pathologiques, assez fréquents chez les vieillards, tels la cholécystite chronique, la décompensation cardiaque, ne sont pas sans affecter l'appétit, la digestion et l'absorption.

Nous pouvons affirmer qu'une diète insuffisante ou mal balancée conduit à un état de nutrition inadéquat à plusieurs points de vue, y compris les vitamines et les sels minéraux. A part cela, il est expérimentalement démontré que, en dépit d'une alimentation suffisante en quantité et en qualité, le vieillissement prédispose à une déficience vitaminique. Chez le vieillard, les réserves sont limitées et le moindre stress peut les brûler rapidement (25).

Quelles sont les déficiences alimentaires chez le vieillard?

La qualité de la diète est aussi importante que le nombre de calories qu'elle contient. Elle doit inclure une quantité convenable de protéines et un surplus plutôt qu'un minimum de sels minéraux et de vitamines. La déficience de protéines est fréquente chez le vieillard probablement à cause de sa privation de protéines dans son alimentation. Une déficience prononcée de protéines est un facteur important dans la perte des tissus, l'anémie, les oedèmes et une déficience moins prononcée peut se manifester surtout par de la fatigue.

Une enquête sur la diète des vieillards a révélé que 26 à 40 pour cent des vieillards, surtout ceux qui pourvoient à leur alimentation, ont un apport insuffisant en protéines (26) (27). Un déséquilibre azoté peut survenir même si l'apport des protéines est suffisant (28); chez des vieillards de 65 à 89 ans, logés et nourris dans des institutions, on a trouvé que les protéines totales plasmatiques étaient normales mais que les albumines étaient inférieures et que les globulines étaient supérieures à la normale et que le rapport serine-globuline était inverse chez quelques-uns (29).

Les déficiences en sels minéraux et en vitamines sont encore plus fréquentes. L'étude de Kaplan (27) a démontré que, chez les individus de plus de 60 ans,

75 pour cent étaient déficients en fer et en thiamine, 60 pour cent étaient déficients en calcium et 50 pour cent l'étaient en vitamine A, riboflorine, niacin et acide ascorbique. Chez un groupe semblable, Jordan (26) a trouvé un apport inadéquat en vitamine A chez 59 pour cent, en acide ascorbique chez 40 pour cent, en calcium chez 43 pour cent.

Un bon état de nutrition aide à conserver les vieillards en bonne santé, les rend capables de mieux supporter le stress de la maladie et de la chirurgie. Par contre, un apport insuffisant en vitamines, surtout en vitamine A, Niacin et acide ascorbique, semble prédisposer le vieillard à un taux de mortalité plus élevé (30).

La diète chez le vieillard

Si l'on ne tient pas compte des modifications apportées par le vieillissement ni des maladies chroniques affectant le métabolisme, les exigences alimentaires des vieillards ressemblent à celles des adultes plus jeunes (31). Toutefois, certaines difficultés empêchent l'institution d'une diète appropriée: des habitudes de vie, des difficultés de la mastication, des problèmes financiers, des facteurs psychologiques. Il s'impose, en conséquence, d'évaluer à fond l'état de nutrition de chacun des malades et de faire, par la suite, des recommandations diététiques individuelles.

Un vieillard en bonne santé a besoin quotidiennement d'à peu près la même quantité de protéines qu'un jeune homme, c'est-à-dire un gramme par kilo de poids. Toutefois, si l'équilibre nitrogéné penche vers l'insuffisance, l'apport de protéines peut être élevé jusqu'à deux grammes par kilo par jour.

Toute perte inaccoutumée de protéines, qu'il s'agisse d'albuminurie ou de drainage, doit être compensée par un apport additionnel de protéines. Les meilleures sources sont: le lait, le fromage, les viandes, les œufs, les céréales et le pain. Le lait écrémé peut être substitué au lait complet si l'on désire restreindre l'ingestion de gras. Le lait écrémé en poudre et les hydrolysats de protéines peuvent également être employés si l'on veut augmenter l'ingestion de protéines sans augmenter trop le volume du repas. Diminuer les graisses pour éviter les troubles gastriques, l'obésité et l'athérosclérose, augmenter le calcium dont l'absorption et l'utilisation deviennent difficiles chez les vieillards et combler les déficiences en fer et en vitamines, voilà les facteurs à se rappeler en prescrivant une diète chez les vieillards.

Robert Elman (11) fait un parallèle entre la perte de poids chez le vieillard et la diminution de sa masse sanguine: cette diminution, dit-il, affecte autant la masse globulaire que les protéines plasmatiques. Au début de 1955, en Angleterre, Ronald W. Raven (14) a fait une étude sur les octogénaires; voici les déficiences les plus fréquentes qu'il a dépistées chez les sujets étudiés: d'abord, une alimentation insuffisante qu'il attribue en partie à des diètes restrictives, en partie à une assimilation défectueuse, ensuite, un déséquilibre de l'hydratation et de la balance des électrolytes, en troisième lieu, une avitaminose.

En 1952, à Boston, Richard Chute (16) s'exprimait ainsi sur la même sujet: "L'amaigrissement chez les vieillards entraîne rapidement un état de dénutrition qui affecte le volume sanguin primitivement dans sa masse plasmatique; viennent

ensuite l'anémie, l'hypoprotéinéhémie avec ou sans oedème, la déshydratation, l'avitaminose et, si l'amaigrissement est plus prononcé, survient un déséquilibre du pH sanguin et de la balance des électrolytes."

En 1954, Julia A. Shea (22) a publié une étude sur les aspects de la nutrition chez les vieillards; elle vient à la conclusion que, au-dessus de 50 ans, le métabolisme basal peut diminuer de 3 à 5 pour cent par décade. Elle a également noté une diminution de la taille et du poids, une diminution des activités corporelles et une diminution des diètes quotidiennes. Un grand nombre de chercheurs ont concentré leurs études sur les déficiences du système cardio-vasculaire chez les vieillards. Joseph Fazekas et ses associés ont étudié, chez l'humain à différents âges, le débit sanguin cérébral et la consommation d'oxygène; ils ont trouvé, chez l'individu de 50 ans et plus, une résistance vasculaire augmentée par l'artériosclérose et une diminution de la consommation en oxygène.

En 1953, P. Schemberg (19) a publié une étude sur la relation entre l'âge, la circulation cérébrale et le métabolisme cérébral; il en est venu aux mêmes conclusions que Fazekas. D'autres auteurs dont Alice N. Bessman et Ass. (23) ont étudié les effets de l'hypotension sur l'hémodynamique cérébrale et le métabolisme du vieillard dès l'installation de l'hypotension, avant même que l'utilisation de l'oxygène soit diminuée; ils ont observé des symptômes qu'on rencontre habituellement dans l'hypoxie, à savoir: nausées, vomissements, instabilité émotionnelle, des modifications de la respiration pouvant aller jusqu'au Cheynes-Stokes.

En interprétant ces observations, les auteurs émettent l'opinion que, chez les vieillards, il existe probablement une incapacité de compenser l'hypotension soit par une vasoconstriction ou une vasodilatation. Toutefois, ils ont noté qu'à la suite de l'inhalation d'une atmosphère contenant 5 pour cent de CO_2, il s'ensuivait chez les vieillards une augmentation du débit sanguin et une diminution de la résistance vasculaire dans l'ordre de 35 à 75 pour cent.

Chez les vieillards, d'après C. W. Semisch (21), le cœur possède moins de réserves où il peut puiser et il devient plus sensible aux stimulations vagales; en plus, soit à cause d'hypertension, soit à cause de maladies vasculaires ou d'artério-sclérose, le cœur, chez le vieillard, est augmenté de volume et il existerait une disproportion entre le débit cardiaque et la demande tissulaire.

Certains pourraient être tentés de mettre en doute l'évaluation clinique des malades. Cependant, d'après Harry Poliakoff (10), elle est assez exacte; il a pratiqué, sur des personnes de plus de 60 ans, 800 post-mortem et il a trouvé que seulement 5 pour cent des sujets étaient exempts de maladies; ensuite, il a comparé les résultats de son examen nécropsique et ceux de l'évaluation clinique, incluant les rapports d'examens radiologiques, électrocardiographiques et autres et il conclut que l'évaluation clinique est exacte.

Quelles sont les opinions sur la tolérance du vieillard à la chirurgie?

Harry A. Oberhelman (2) prétend que, chez le vieillard, toutes les maladies requérant de la chirurgie prennent une importance majeure. Actuellement, toutefois, la compréhension plus complète des changements physiologiques du vieil âge, la collaboration de médecins internistes, de chirurgiens et d'anesthésistes plus

entraînés ont considérablement réduit le risque de la chirurgie majeure chez le vieillard. Le dicton: "Il est trop vieux pour être opéré" ne devrait plus se répéter aujourd'hui.

Après avoir cité les élogieuses paroles de Cannon sur l'anesthésie moderne, Thomas J. Anglin (15) s'exprime ainsi: "Un des facteurs qui a le plus contribué à réduire le risque opératoire chez les vieillards est le perfectionnement de l'anesthésie. L'extrême vulnérabilité des vieillards à l'hypoxie et à l'hypotension exige que l'anesthésie soit administrée par un anesthésiologue qui possède une grande connaissance de la physiologie respiratoire et circulatoire et qui a, en plus, à sa disposition, les moyens de prévenir ces dangereuses complications."

Au sujet de la thérapeutique chirurgicale chez le vieillard, le professeur Henri Simonnet (20) s'exprime ainsi: "C'est surtout dans le domaine de la chirurgie des personnes âgées, que les progrès les plus intéressants ont été réalisés. Ces progrès résultent moins de l'amélioration de la technique chirurgicale en général que dans la mise au point de méthodes appropriées à la préparation du sujet à l'anesthésie, aux soins donnés durant l'opération et dans la phase post-opératoire. Ces méthodes n'ont pu être élaborées que grâce à une connaissance plus approfondie de la physiologie de l'organisme âgé et des modalités de ses réactions à l'acte opératoire."

L'auteur énumère ensuite les principales modifications sur chacun des systèmes, modifications dont il faut tenir compte au cours de l'anesthésie.

Préparation à l'opération

Il est fortement conseillé, sinon unanimement conseillé par les auteurs, de faire, avant l'opération, une évaluation soignée des différents systèmes du malade: évaluation clinique, tests de laboratoires, électrocardiogramme, examens radiologiques, etc.

Une fois renseigné sur les points faibles de son malade et sur ses plus sérieuses déficiences, l'anesthésiste commencera, le plus tôt possible, à prescrire les traitements nécessaires pour rapprocher le malade le plus près possible de la normale avant l'opération. Ensuite, l'anesthésiste devra évaluer les exigences de l'opération selon son site, sa durée, selon l'importance du traumatisme, les pertes sanguines, le déséquilibre hydrique et électrolytique, selon la durée du jeûne post-opératoire, la durée de l'immobilisation, l'inhibition de la respiration, de la circulation, etc. . . . Cette évaluation faite, l'anesthésiste augmentera en conséquence les réserves du futur opéré. Il est recommandable de laisser les malades actifs, de leur donner une diète riche en protéines et en vitamines, un grand nombre de calories sous n'importe quelle forme, même de l'alcool.

Il ne faut pas hésiter à recourir aux transfusions pre-opératoires, mais il faut donner de préférence de petites transfusions quotidiennes et du sang total et frais.

Le dosage des ingesta et des excreta et la recherche de l'équilibre des divers électrolytes sont toujours utiles chez les vieillards.

Le professeur Henri Simonnet, dans ses cours sur la nutrition chez le vieillard, donne de judicieux conseils sur l'évaluation clinique de l'état de nutrition et sur les tests utiles dans ces circonstances. Avant l'opération, il faut, chez le vieillard

encore plus que chez les autres malades, renseigner le malade sur les exigences post-opératoires: les activités interdites et les activités obligatoires; lui montrer à respirer, à tousser, à faire du drainage postural; le renseigner sur son alimentation et son hydratation interdites ou prescrites.

Quand arrive le moment de la prémédication chez les vieillards, il faut éviter tout ce qui pourrait entraîner une dépression centrale: tout ce qui pourrait diminuer les échanges respiratoires ou faire baisser la tension artérielle est susceptible de diminuer l'oxygénation des tissus; puis, chez le vieillard, il est strictement interdit de tolérer la moindre hypoxie.

En maintes occasions, une bonne psychothérapie peut réduire de moitié la prémédication ou même la remplacer entièrement.

Au cours de l'opération, il y a deux choses qu'il faut éviter en tout temps: c'est le manque d'oxygène et les chutes de la tension artérielle. Le sang perdu doit être calculé et remplacé précocement en qualité et en quantité; plusieurs auteurs conseillent d'en donner plus que moins. Une opération lente et douce, selon l'avis de plusieurs, demeurerait moins choquante qu'une opération courte et traumatisante.

En ce qui concerne les agents anesthésiques généraux, le cyclopropane et l'éther retiennent les préférences de la plupart des anesthésistes; les associations d'agents sont également conseillées mais l'usage du protoxyde d'azote ne semble pas souhaitable car le mot d'ordre demeure: assurer une oxygénation abondante et éviter les dépressions. Parmi les facteurs pouvant influencer le choix des agents anesthésiques, il faut mentionner: la durée de l'opération, la profondeur de l'anesthésie requise, la gravité des pathologies cardio-vasculaires et respiratoires du malade et, enfin, l'expérience de l'anesthésiste à administrer ces agents.

La majorité des anesthésistes expérimentés semblent opter pour l'intubation trachéale chez le vieillard, surtout s'il est porteur de sécrétions bronchiques. La toilette de la trachée et des bronches au cours de l'opération et particulièrement à la fin de l'opération peut permettre une meilleure oxygénation et éviter, chez des vieillards dont la musculature striée est atrophiée, des efforts répétés et épuisants pour expulser quelques sécrétions dont l'accumulation pourrait les conduire à l'atélectasie, à la pneumonie et même à l'asphyxie.

Chez le vieillard, il faut réduire les solutés intra-veineux comparativement à l'emploi qu'il en est fait chez les autres malades; il en est ainsi de l'usage des liquides à pression osmotique faible; de l'avis du plus grand nombre, au cours de l'opération, il vaut mieux hypohydrater le vieillard que de l'hyperhydrater.

Après l'opération

Chez le vieillard tout particulièrement, il faut encourager le lever et la reprise précoces des activités normales. Une alimentation à haute teneur en calories doit également être reprise précocement. Il faut exercer une surveillance continuelle sur le système respiratoire des vieillards, encourager les exercises respiratoires et le drainage postural et ne pas hésiter à pratiquer et à répéter des aspirations bronchiques. La sédation doit être variée et ne jamais entraîner de dépression. La circulation doit être maintenue active, la masse sanguine être évaluée fréquemment et être maintenue adéquate par des transfusions.

Résumé

Les statistiques laissent entrevoir une augmentation plus rapide du nombre de vieillards que du reste de la population. Conséquemment, il est à prévoir que le nombre de vieillards devant subir des interventions chirurgicales augmentera proportionnellement.

Nous avons mis en évidence les déficiences les plus fréquentes qui accompagnent le vieil âge. Nous avons donné quelques moyens d'augmenter les réserves et la résistance des vieillards et, du même coup, leur tolérance à la chirurgie.

Ce qui est de rigueur d'éviter en tout temps chez les vieillards, c'est l'hypoxie et les chutes prononcées de tension artérielle. Nous avons fait des suggestions pour éviter ces complications au cours de l'opération et durant les suites opératoires. La mise en pratique de ces conseils, nous en avons la conviction, peut encore diminuer le risque opératoire, la morbidité et la mortalité chez les vieillards soumis à la chirurgie.

Summary

In all countries, statistics show that the number of aged persons is increasing faster than the rest of the population. Consequently, it is evident that more and more elderly persons will be brought to surgery and anaesthesia.

The authors have discussed the multiple deficiencies occurring in old age. Suggestions have been made for treatment which will increase the reserves and resistance to surgery of the aged.

It is mandatory in the aged to avoid hypoxia and hypotension. The authors have made suggestions for the prevention of these complications, both during and after operation. By proper treatment it is possible to decrease morbidity and mortality in the elderly patient coming to surgery.

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THE COMPLICATIONS OF ENDOTRACHEAL ANAESTHESIA*

H. C. Carruthers, M.D. and H. B. Graves, M.D.**

Gillies points out that of all the technical improvements designed to make inhalation anaesthetics safer, simpler, and more efficient, tracheal intubation is one of the most notable (1). Its advantages were recognized by Trendelenburg, McEwen and others, who realized the need for an unimpeded channel for anaesthetic vapours and oxygen, and simultaneous protection of the unconscious patient against aspiration of blood and debris during operations on the mouth and pharynx. Need for a near perfect airway has become even more important since the introduction of methods involving generalized muscular paralysis, where ventilation has to be maintained by the anaesthetist. Surgeons and anaesthetists know that accidents occasionally occur in the performance of difficult techniques. These reports should not discourage us from an undeniably useful procedure, but should help us avoid some of these complications. The discussion today concerns itself with these complications.

Trauma during intubation is a fairly common occurrence; it is more frequently associated with the inexperienced anaesthetist (2). Insertion of a laryngoscope may result in lacerated lips, abraided gums, loosening, fracture or extraction of teeth, abrasions and lacerations of the hard and soft palate and the posterior wall of the pharynx, and damage to the tongue. Insertion of a tube through a nostril may cause nasal mucosal abrasions, especially in the region of the turbinates and anterior septum, with resultant epistaxis (3, 4). A nasal polyp may be traumatized, or even avulsed with resultant bleeding.

During intubation the supraglottic, glottic, or infraglottic structures may be injured. These may be soft tissue injuries, or cartilage and perichondrial injuries. The soft tissues of the membranous glottis may sustain minimal abrasion or crushing. Recovery is usually prompt. A puncture injury of mucosal and submucosal tissue may result in ecchymosis, hematoma, or even emphysema. Excoriated areas may become infected and covered with an exudate, which can lead to formation of a granuloma. More extensive trauma to the membranous glottis may be a laceration, or even avulsion of a cord. Forward elevation of pharyngeal structures during intubation causes a stretch, resulting in transient pharyngeal discomfort.

The most common reaction following intubation, usually seen in the first 24 to 72 hours, consists of loss of lustre, slight rounding, and pinkish infiltrations of the vocal lips of the true cords. Trauma of a more severe degree, or even minimal trauma with added infection, may result in marked inflammation and acute laryngeal obstruction.

Cartilage and perichondrial injuries may involve the epiglottis, thyroid, aryte-

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**Department of Anaesthesiology, Vancouver General Hospital; University of British Columbia, Vancouver, B.C.

noid or cricoid cartilage, or the crico-arytenoid articulation. Inflammation of the lingual surface of the epiglottis may cause oedema, or even abscess formation. The swelling does not spread onto the laryngeal surface, as the mucous membrane there is tightly adherent.

The reparative process for cartilage in the larynx is very poor. If cartilage and its perichondrium are injured, perichondritis, prolonged suppuration, and cartilaginous necrosis with scarring and deformity result. Laryngeal stenosis ensues if remedial measures are not taken to prevent it. In general, most intra and extra laryngeal damage fortunately consists of slight mucosal engorgement, with little if any subjective complaints, or functional disturbances. In many cases of sore throat there is little or no evidence of trauma. Conversely, positive evidence of trauma does not always produce symptoms. Wylie says that in view of the vast number of intubations, laryngitis is relatively uncommon (5).

Acute oedema of the glottis may follow intubation (8). It is more common in infants than in older children or adults. Crispell and L. J. Hampton in reviewing 337 children receiving endotracheal anaesthesia over a 18-month period, beginning October 1947, at the Grace New Haven Community Hospital, reported that there were two cases requiring tracheotomy for laryngeal oedema (6). In those two cases the cerebellar head rest had been used. Mild degrees of laryngeal dysfunction with hoarseness were fairly common. Shaw reports a case where a 25-year-old burned negro patient, who had had a difficult induction and intubation, and four and one half hours of skin grafting with the head in a cerebellar rest, became completely obstructed on extubation, so that tracheotomy was necessary. They were unable to re-insert the tube as the cords were not visualized, and the aryepiglottic folds were seen to be a pinkish mass of oedematous tissue (7). In most of the cases of severe oedema reported, it appeared that induction and intubation were usually lengthy and difficult.

Gillespie attributes laryngeal oedema to a streptococcal infection, which gains entrance into a mucosal abrasion at the time of operation (8). Mechanical irritation plays a role. Elevation of a cranial bone flap, for instance, necessitates a considerable movement of the head and neck, with possible irritation of structures adjacent to the larynx. As to etiology of tracheal and laryngeal oedema in children, R. M. Smith mentions the following causative factors: oversized tubes and laryngoscopes, intubation during light anaesthesia and spasm, unstable apparatus dragging on the tube, a tugging respiration see-sawing the tube back and forth across the cords, high flows of unhumidified oxygen, and the improper cleansing and storage of tubes, scopes, and catheters (9). Deming, in reporting on anaesthesia for 49 cases of tracheo-esophageal fistula in the newborn, and on 250 other intubations of patients under fourteen days old, had no cases of obstruction from laryngeal oedema. All these patients were placed in an isolette with humidified oxygen after operation. She believes that absolute cleanliness of tubes together with correct size, and atraumatic intubations, are the most important factors in avoiding laryngeal oedema (10).

Many cases of laryngeal granulomata following intubation have been reported. Gillespie states that these may follow use of many types of tubes, even of the softest variety, and may ensue after either nasal or oral intubation (8).

Young and his colleagues at the Manchester Royal Infirmary recently report an interesting series of twelve cases seen in three years of routine E.N.T. practice (11).

A few days after the anaesthetic, the appearance is that of an acute laryngitis, but when this general inflammation subsides, a small area of ulceration may be seen at the glottis, usually on the vocal process of one or both arytenoid cartilages. Granulations may proliferate at the ulcer site, and may organize to form polyps, or a pedunculated fibroma in the course of time. Apparently a similar nodule may form as a submucosal hematoma. Grossly these lesions vary in size, shape, and position. In most of their cases, the polyps appeared on the posterior one-third of the cords. Not infrequently the site is in the interarytenoid area, just below the glottis. In either case there is a tendency to web formation later. The microscopic picture usually shows a small nodule with thickened epithelium, superficial ulceration, and subepithelial haemorrhage.

Some cases are symptomless, and would only be picked up on routine laryngoscopy. The commonest symptoms encountered were hoarseness, intermittent loss of voice, dyspnoea, pain or discomfort in the throat, rarely haemoptysis in the first few weeks; later there may be a sensation of something moving in the throat if a pedunculated polyp is present. The condition is essentially one of adults. Acute oedema of the larynx in children seems to be the corresponding effect of the lax tissues of childhood. Granulomatous lesions are more frequent in women than in men.

Etiology includes injury on passing the tube and movement between the tube and cords. Pressure by the tube is considered important by Dwyer and his colleagues, in causing tracheitis and granulomas (12). They demonstrated that the curve of the tube may force it into the back of the glottis, and at the same time against the front of the trachea. They suggested a double curved tube, so moulded as to fit as nearly as possible into the contour of the pharynx, larynx, and trachea, when the head and neck are in normal relationship to each other. Young *et al.* could show no relationship between these lesions and the materials of which the tube is made, or between them and any particular antiseptic or lubricant used (11).

Regarding prevention, these authors stress gentle respiration, when the tube is passed, afterwards, and until it is withdrawn. Dwyer *et al.* say that raising and flexing the head lessens the curve the tube must take at the larynx, and hence lessens the pressure thereon. Lowering and extending the head increases the angle of the tube at the cords, and hence the pressure. On the other hand R. T. Barton advises that one should avoid a sharply flexed head position, and strive for hyperextension (13).

Young *et al.* say that if an ulcer occurs, complete voice rest will help it to heal without formation of excessive granulation tissue. There is a tendency for nodules to disappear naturally, and removal should be postponed until it is apparent that this is not going to take place. Symptoms may eventually dictate the need for excision, simple snaring or punching out by direct laryngoscopy being sufficient. However, the operation may have to be repeated, and this should be done before too much time elapses.

A pathological entity termed "subglottic membrane" has been reported by

Etsten and Mohler (14), Baron and Kohlmoos (15), Snyder and Gants (16), and Muir and Stratton more recently (17). Etsten and Mohler had three cases out of eight hundred adult intubations. These three developed acute upper respiratory obstruction 24 to 48 hours after anaesthesia. Clinically there was cough, dyspnoea, and suprasternal retraction. On direct laryngoscopy, the glottis, epiglottis, and larynx appeared normal, and there was no laryngeal oedema. Bronchoscopy revealed an adherent subglottic membrane. This could have been missed if a tracheotomy had been done to relieve obstruction before an accurate diagnosis was made. Etsten's three cases, all were women with short fat necks. Magill tubes with cuffs, inserted with a stylet, were used. The tubes all fitted snugly, and the cuffs were therefore not inflated. A sense of resistance was encountered during extubation.

The subglottic area is the narrowest portion of the larynx, completely surrounded by the cricoid cartilage with mucosa attached. This area is very prone to injury with subsequent infection. A slight narrowing of the lumen here easily results in obstruction. The mucosa may be damaged by the tip of the tube, the point of a stylet, or by prolonged pressure of a deflated or wrinkled cuff. The use of a small smooth cuff on a tube properly sized, with the cuff placed entirely below the cricothyroid mucosa, eliminates this factor.

After injury to the subglottic mucosa, leukoplakia and desquamated epithelial cells accumulate about the abraded area, and a fibrinous exudate forms, adhering to the anterior wall of the cricothyroid membrane. The medial aspect of this formed membrane, when free, comes in contact with the inferior surface of the vocal cords causing obstruction.

Differential diagnosis of obstruction can be greatly aided by direct laryngoscopy. Laryngeal oedema, granuloma of the vocal cord, or a foreign body can be seen by this procedure. If the glottic and supraglottic areas appear normal, bronchoscopy may reveal a subglottic membrane, slough of bronchial mucosa, or necrotic tracheitis. Treatment of subglottic membrane is immediate removal by bronchscopy. If the entire membrane is not removed it may recur in 24 hours.

Tracheitis is a definite entity associated with retrosternal soreness, and usually some cough (5). F. L. Turner has described three cases of slough of tracheal mucosa. This may cause respiratory obstruction within 24–48 hours, characterized by a high-pitched wheeze. The obstructive signs appear mainly in the expiratory phase of the respiratory cycle (18).

Necrotic tracheitis is a rare and terrible complication. Ransom describes a case where a 37-year-old woman had a myomectomy under a pentothal, cyclopropane ether anaesthetic, using a #7 Magill tube (19). Next day the patient became dyspnoeic, with some stridor and production of green sputum. On the third day tracheotomy was performed with great difficulty but the patient died on the table. Necropsy showed the larynx and trachea smaller than normal, with a subchronic inflammation in the trachea and bronchi, with superimposed acute changes in the trachea, bronchi, and lungs. The tube was made of war-time rubber, and had been soaked in 1:1000 biniodide of mercury. Patch tests on volunteers using various combinations of rubber, cotton, and solution seemed to incriminate the mercury solution, and thereafter no tubes were soaked in it.

The major postoperative respiratory complications include pneumonia, acute

bronchitis, empyema, lung abscess, and atelectasis (8). Almost all are infective. These are held responsible for a large number of post-operative deaths. They are especially common with patients in poor physical condition. Bronchopneumonia is a common post-mortem finding in all elderly patients. It is equally common in patients who die after operations. In the latter case, the complication was previously labelled "ether pneumonia." It is now believed that the pulmonary lesion is only indirectly attributable to the effects of the anaesthetic, and that the type of operation performed, and the condition of the patient, are important causative factors. Campbell and Gordon surveyed postoperative complications in 2094 unselected anaesthetics, and found that the incidence of complications was less in the group who were intubated (20). They concluded that this was probably owing "to the exclusion from the trachea of infection, and secondly to the opportunity presented to the anesthetist for removing mucus from the trachea by suction through the tube." There is, of course, a possibility of introducing infection from the nose, and many spirited articles have been written regarding this possibility. One surgeon was quoted as describing nasal intubation as a "perfectly filthy method." It is probably the feeling of most modern anaesthetists that unless there is a definite indication for nasal intubation, the oral route is to be preferred.

Cardiac arrythmias often appear during intubation and extubation. Burstein has reported on this complication, attributing them to insufficient depth of anaesthesia, prolonged laryngoscopy, respiratory obstruction, and irritation of the tube. In another article he points out that most of the arrythmias are transient, and of no grave importance (21, 22).

Finally, mention will be made of the rarer complications. Wylie reports a case as follows (5). An 18-year-old boy developed a laryngospasm on induction. This was finally controlled, he was intubated, and the pharynx packed. At that time some engorgement of the larynx and pharynx was noted. In ten minutes there was fluid in the tracheal tube. This was sucked out. After operation, he was extubated, but laryngospasms and cyanosis recurred. A diagnosis of acute pulmonary oedema was made; an orotracheal tube was again passed, and much pink frothy fluid aspirated. During the next 45 minutes his condition deteriorated in spite of cardiac stimulants and oxygen, and he succumbed. Post-mortem examination showed the lungs were solid with haemorrhagic fluid, with uniform oedema throughout. The heart showed a dilated right ventricle. The meninges, brain, and liver were engorged.

Barnard reports an unusual incident during nasal intubation. When the tube reached the laryngeal region, a sudden resilient resistance was felt. On direct laryngoscopy it was noted that the tube was covered with pharyngeal mucosa. He had entered the submucosa in the nasopharynx, and had passed the tube down under it (4).

Finer reports a case where an unsuspected mucous cyst of the right arytenoid process of the larynx caused no obstruction until after pentothal and curare were given. This resulted in such distortion that it was not possible to intubate until muscle tonus had returned. Some oxygen was finally forced in when the patient's head was turned to the right. Thus it must be recognized that distortion of

anatomical relationships by anaesthesia may precipitate a mechanical obstruction in a patient who otherwise can maintain a patent airway while awake (23).

Impaction of the epiglottis has been reported (24). In this case when the second plane of surgical anaesthesia was attained, obstruction accompanied by a strange flapping noise occurred. On laryngoscopy it was noted that an overhanging epiglottis had been sucked down between the cords, causing a valve type obstruction. Intubation remedied the trouble.

It cannot be too strongly emphasized that insertion of an endotracheal tube with too small a diameter may cause obstruction, especially in children and infants. An aged tube may kink in the region of the pharynx. R. W. Shaw points out that a tube may become solidly plugged with blood or secretions causing the very thing it was meant to prevent (25).

Cuffs on tubes may cause obstruction. Too thick a cuff will require so much pressure for inflation, that a Magill tube may be collapsed by it. Too thin a cuff may bulge out in front of the tube, especially if over-inflated, causing a condition resembling bronchospasm. The remedy is to release the cuff (8). Loss of a cuff from a tube may occur. Lennon and Rovenstine reported a case in which death followed the rupture of an inflatable cuff inside a trachea which was already damaged (26). The patient developed subcutaneous emphysema, and died on the second day after operation. Autopsy showed a break in the mucosa of the right main bronchus, in an area of long standing ulceration. This accident was due to two rare phenomena, rupture of a cuff and ulceration of a bronchus.

We are all familiar with the possibility of intubating the right main bronchus and the trouble it may cause. Bishop reports a case in which an 11-month-old infant was intubated for cheiloplasty using a makeshift tube cut from a #16 French catheter (27). Almost immediately respirations ceased. Inflation of the lungs was attempted. Death ensued in one or two minutes in spite of resuscitative efforts. At necropsy, the respiratory tract was removed in one piece. It became apparent that the tube was placed down the right main bronchus, at the same time shutting off the bronchial division of the left lung, and a portion of the divisions of the right lung. It had not been appreciated that an infant's trachea is only 4 cm. long. Leigh and Belton claim that this difficulty is further enhanced since children are diaphragmatic breathers, and this causes a slight ascent and descent of the trachea on inspiration and expiration (28).

There are, of course, many dangers associated with using compressed gases in endotracheal anaesthesia. The pressure in a full cylinder varies from 2000 lb. to the square inch in the case of oxygen, to 75 lb. for cyclopropane. If by some accident such pressures are brought to bear on the patient's respiratory tract, grave damage will ensue. Even reducing valves may be set up to 80 lb. per square inch. The danger to the patient of an accidental release of a gas is greater when endotracheal anaesthesia is in use. Experimentally, Marcotte *et al.* have shown that an intrabronchial pressure of 24 mm. Hg. produced mediastinal emphysema in dogs (29). The danger of causing emphysema, or even rupture of the lungs in a human, is obvious. Needless to say, if an explosion occurs, the danger to the patient is greatly enhanced if he is intubated, and is on a closed or semi-closed system.

Loss of a tube below the cords may occur. Paralysis of the vocal cords following endotracheal anaesthesia is in most instances due to inadvertent section of the recurrent laryngeal nerves. Trauma to the carina has been reported, as well as tracheal webs, leucocytic membrane of the end of the trachea, and perforation of the trachea. Tubes have been inadvertently passed into the stomach, and that viscus ruptured by being forcibly filled with oxygen or gas. Rupture of an aortic aneurysm is considered to be a possibility during intubation, although I have not read of such an accident occurring.

Summary and Conclusion

I have attempted to deal with the complications of endotracheal anaesthesia, stressing some and of necessity dealing briefly with others. These sequelae include injuries and infections of the supraglottic, glottic, and infraglottic areas. Special mention has been given to such entities as laryngeal oedema, laryngeal granuloma, subglottic membrane, and laryngotracheitis. The more unusual complications connected chiefly with mechanical difficulties have been considered. In most instances recommendations for their prevention or treatment have been outlined.

In spite of these complications, the merits of endotracheal anaesthesia stand established at the present time. Sometimes undesirable features are associated with its management, and, unfortunately, these always accrue to the patient. Careful discrimination in regard to the selection of the technique, and meticulous management when it is chosen, are essential safeguards in its application.

Résumé

Parmi tous les perfectionnements techniques destinés à rendre l'anesthésie par inhalation plus sûre, moins compliquée et plus efficace, l'intubation endotrachéale est l'un des plus importants (1). On lui reconnaît les avantages suivants: elle procure une voie libre pour les vapeurs anesthésiques et l'oxygène en même temps qu'elle protège le patient inconscient contre l'aspiration de sang et de débris au cours des interventions sur la bouche et le pharynx. Des accidents surviennent quelquefois au cours de manipulations présentant des difficultés techniques. Leur éventualité ne doit pas nous empêcher cependant de pratiquer cette technique d'une utilité indéniable mais elle devrait nous aider à éviter quelques-unes de ces complications.

Au nombre des traumatismes pouvant survenir au cours de l'intubation, on peut mentionner des coupures aux lèvres, des éraflures aux gencives, des lésions aux dents ou à la langue, des éraflures et lacérations au palais dur ou mou ainsi qu'à la paroi postérieure du pharynx. L'intubation nasale peut causer des éraflures à la muqueuse ou l'arrachement d'un polype avec épistaxis consécutif. Les structures supraglottiques, glottiques ou infraglottiques peuvent subir des lésions susceptibles d'entrainer par la suite la formation d'ecchymoses, d'hématome, d'emphysème ou d'un granulome. L'arrachement d'une corde voccale peut se produire. Une sténose de la glotte peut survenir par nécrose post-traumatique d'un cartilage et infection consécutive.

Dans plusieurs cas de maux de gorge à la suite d'intubation, on ne retrouve

que peu ou pas d'évidence de traumatisme, et, par contre, le traumatisme ne produit pas toujours des symptômes.

L'intubation peut être suivie d'un œdème laryngé, de granulomes ou de polypes du larynx. Ces troubles seraient causés soit par le traumatisme à l'introduction du tube, soit par les mouvements des cordes vocales sur le tube. On rapporte de l'obstruction respiratoire aiguë à la suite du développement d'une membrane sous-glottique après intubation (14, 15, 16, 17).

Une trachéite peut se développer et causer de l'obstruction respiratoire endedans de 24–48 heures. Une trachéite nécrosante est une complication rare mais terrible (19).

Des arythmies cardiaques se produisent souvent durant l'intubation ou à l'extubation. La plupart de ces arythmies sont transitoires et d'aucune gravité; on les attribue soit à un niveau d'anesthésie trop léger, soit à une laryngoscopie prolongée, ou à de l'obstruction respiratoire ou à l'irritation causée par le tube (21, 22).

L'auteur discute aussi des facteurs mécaniques qui peuvent produire une obstruction respiratoire durant l'anesthésie endotrachéale.

En dépit de ces complications, les avantages de l'anesthésie endotrachéale demeurent bien établis à l'heure actuelle. Son emploi s'accompagne de temps en temps d'effets indésirables et malheureusement ils sont toujours au détriment du malade. Il faut user de discernement dans le choix de la technique et, quand le choix est fait, ne faire que des manipulations de toute délicatesse: voilà les gages de sécurité de son emploi.

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THE ROLE OF DRUGS IN FOETAL MORTALITY*

NEIL A. STEWART, M.D.**

IT HAS BEEN SAID that the most dangerous day of our life is the day on which we are born. There is no doubt that this statement is true, and while there have been great advances in our modern age in pre-partum care, antisepsis, method of delivery and infant care, there have been added during the past one hundred years additional hazards attendant upon the use of obstetrical analgesia and anaesthesia.

Since Queen Victoria received chloroform in 1853 at the birth of Prince Leopold, the use of pain-relieving drugs has become an established procedure. It is only during the present century that the use of analgesic drugs in the first and second stages of labour has become popular. In 1902 Von Steinbuchel added scopolamine to morphine and so the famous era of "Twilight Sleep" was born. This combination was used very extensively for about twenty years and was followed by many other drugs, such as large doses of paraldehyde by rectum, tremendous doses of barbiturates by mouth, and a number of others. Many of these drugs have been discontinued because of their obvious effects on the foetus, and we are now in the stage where we are using not only a few of the old and trusted drugs but also certain new drugs and combinations of drugs which are felt to be safer for the infant.

It is extremely difficult to evaluate the effects of analgesia and anaesthesia on the foetus in a scientific manner, and it is necessary to rely to a great extent on clinical impressions at the time of delivery, the history of the baby following delivery, or autopsy findings in the case of the stillborn or those dying soon after birth. However, a great deal of experimental work has been done involving the use of animals during the past twenty years, and while it is always dangerous to assume that animals react in the same way as human beings nevertheless this work can at least give us a lead as to what is happening in the case of humans.

In order to estimate the effects of drugs on the foetus, it has been necessary to find some way of assessing these effects without the other complications of labour. This has been found to be possible in the case of the rabbit, which comes to full term at thirty days (1). By the administration of corpus luteum material, the gestation period can be prolonged to at least thirty-two days. During this time the foetus can be observed under both normal and abnormal conditions. For instance, the uterus can be opened under conduction anaesthesia in a saline bath and the litter watched under direct vision with the placental circulation intact. A number of very interesting things can be observed by this method, and it can be definitely proved that the foetus breathes amniotic fluid, not only by watching the breathing but by introducing various dyes into the fluid and later finding the dyes in the lungs of the foetus. The effects on the foetus of drugs and anaesthetics administered to the mother can also be observed, and it has been

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**St. Vincent's Hospital, Vancouver, B.C.

shown that some drugs slow the breathing and others completely arrest it. It has also been demonstrated by this method that there are numerous cases of foetal pnuemonia and asphyxia neonatorum that are not related to analgesia and anaesthesia but are present for a considerable time before birth, and that therefore the use of such drugs cannot be held accountable in all cases where there seems to be no logical explanation for the birth of a dead baby.

A great deal has been said and written about sensitivity of the foetal respiratory system to drugs and anoxia. While there is no doubt that this partly exists, the most amazing thing about a foetus is its ability to withstand trauma, narcosis, and anoxia. Every day healthy, lusty babies are born by narcotized, anaesthetized mothers. It is a matter of record that the cord blood at birth shows much less oxygen concentration than that of the mother and that some babies can live for a period of thirty minutes following birth without drawing a breath, which would indicate a resistance to lack of oxygen at least ten times that of an adult.

In order to have an understanding of the effects of drugs on the foetus it is necessary not only to study the direct effects on the foetus but also to study their influence on the mother; whether they tend to increase or decrease the force and frequency of the contractions; whether labour is prolonged or modified by the agent used; the transmissibility of the drug through the placental membrane; the mode of such transmission and the variations in oxygen tension in the mother's blood, all of which factors have a place in determining the final outcome for the foetus. It has been shown, as previously mentioned, that certain drugs and anaesthetics inhibit the respiration of the foetus *in utero* and it has also been shown that babies born in advanced anoxic condition owing to depression of their respiratory activity directly by drugs, or owing to the action of these drugs in prolonging labour, even though resuscitated often remain in a lethargic or deeply depressed condition for days, and in many cases die in the first week of life. It consequently becomes important to know, not only from a standpoint of foetal mortality but also of foetal morbidity just what action the various agents we use have both on mother and foetus and also the best time during labour for administration of these drugs.

In general, the drugs and anaesthetics used during labour act in two ways: (i) they raise the pain threshold, and (ii) they produce a feeling of sleepiness, euphoria and well-being.

Most of the drugs can be classified under the following headings:

(1) Narcotic drugs such as morphine, codeine, pantapon, Demerol and heroin, which act mainly by raising the pain threshold but also produce euphoria.

(2) Barbiturates such as Nembutal, Seconal, sodium pentobarbital, and Pentothal, which act mainly by producing euphoria, drowsiness, and sleep.

(3) Drugs such as scopolamine which produces amnesia.

(4) The anti-histamine group such as Largactil, Phenergan, which produce drowsiness and tend to potentiate the action of other drugs.

(5) Inhalation anaesthetics such as ether, chloroform, nitrous oxide, trichlorethylene, and cyclopropane.

(6) Conduction anaesthetics—Procaine, Pontocaine, Nupercaine, etc

While it is not possible at this time to discuss each drug separately, an effort will be made to discuss them as groups and to indicate the advantages and disadvantages of each group.

As has been mentioned previously, morphine and its derivatives, particularly in combination with scopolamine, were very popular for many years. There is no doubt that this combination has been one of the most effective in easing the pain of the pre-delivery period of child birth. However use of it has been largely discontinued as it has been found clinically to cause too much depression to the foetus, and more particularly so if administered between the second and third hour before delivery.

The practice of using large doses of barbiturates has also been largely discontinued, and these drugs have been blamed for causing lethargic depressed babies who do not breathe well or nurse readily. However, the interest in Sodium Pentothal has recently been revived and there are some anaesthesiologists who are using Pentothal as an anaesthetic for delivery (2). Levis of London has been using this drug for many years (3). His method is to give ¼ gr. Pentothal about twenty minutes before delivery, and he states that while the concentration of Pentothal in the mother's blood and the cord blood is the same at the time of birth, nevertheless he has never noticed any depression of the baby which he felt could be attributed to the anaesthetic. In support of this observation, work done during the past year on humans by McKechnie and Converse of Albany indicates that the foetal blood reaches equilibrium with the maternal blood three minutes after the injection of Sodium Pentothal. They also state that the reason for their favourable findings on the use of Pentothal for obstetrics is that this drug administered intravenously is distributed throughout the body through a fat phase and a water phase, the latter consisting of blood and organ tissue, and that the concentration in blood and organ tissue passes into fat. They therefore think that a large portion of the initial Pentothal injection is removed by the tissues and that not enough is transferred via the umbilical cord to reach a level in the brain of the foetus adequate to produce depression. If these observations are correct, it would appear that a single dose of Pentothal would not be injurious to the foetus but that repeated injections would be, owing to storage of the drug in fat.

Of the volatile anaesthetics and gases in use, ether, nitrous oxide, and cyclopropane are the most common. In work done by Snyder on animals with ether it was found that foetal respiration was completely abolished when the level of surgical anaesthesia was reached in the mother. However ether is nevertheless an excellent anaesthetic for delivery provided it is not administered for too long a time or to the level of surgical anaesthesia, and its safety can be attested by the fact that it is widely used throughout the country, particularly where trained anaesthetists are not available.

Nitrous oxide is a remarkably safe anaesthetic provided that a ratio of 80 per cent nitrous oxide and 20 per cent oxygen is not exceeded. It has very little effect on the foetus, provides satisfactory analgesia for the mother, and is rapidly eliminated.

The so-called conduction type of anaesthetic is probably safest for the baby, with one reservation. It has been noted that if there is too much reduction in

blood pressure of the mother, the relative blood pressures of the mother and foetus are disturbed. This differential blood pressure has been loosely called the "placental blood pressure," and while other factors are involved there is no doubt that a lowering of maternal blood pressure could have a serious effect by altering the passage of gases through the placenta. However, the point is relatively academic and could be readily compensated for by the use of vasopressors and the administration of oxygen to the mother. In spite of the fact that conduction anaesthesia is safest for the baby in that it does not involve the use of narcotic or depressant drugs, it nevertheless enjoys limited popularity. Epidural and caudal blocks are the methods commonly used today. Many dangerous complications may ensue, such as arrest of labour, high or total spinal anaesthesia, or introduction into the spinal fluid of lethal doses due to congenital abnormalities or lack of skill on the part of the operator.

Recently a group of synthetic anti-histamine type drugs have appeared on the obstetrical horizon and have been subjected to extensive clinical tests. These drugs, of which Phenergan and Largactil (4) are an example, are often called "neuroplegic" drugs. They act by producing torpor and somnolence and by potentiating the action of other drugs and anaesthetics. These drugs also have certain side effects such as partial blockage of both sympathetic and para-sympathetic systems, and thus have an effect on maternal blood pressure which is rather unpredictable. Opinions vary regarding the use of these drugs. However one large obstetrical hospital in the city of Vancouver uses Phenergan with very good results and apparently with very little effect on the babies.

Before closing it should be stated that this paper has had to do with full-term babies. It is generally agreed among obstetricians and anaesthesiologists that the premature infant is particularly sensitive to alterations in its physiological functions attendant upon the use of drugs, and that the greatest care should be used in handling these cases. Recently a drug which bears the trade name of Nalline has come into extensive use as an antidote for narcotic over-dosage (5). Many obstetricians wish this drug to be administered either to the mother before delivery, or to the baby after delivery. This is an excellent drug when used in its proper place but it tends to intensify and prolong the action of barbiturates and general anaesthetic agents, and in obstetrics should only be used in those cases where these have not been administered.

Conclusions

1. The best combination of drugs in the medical armamentarium for the reduction of pain during labour has been that of morphine and scopolamine, but this combination has proved very dangerous to the baby.

2. The use of barbiturates is generally contra-indicated, but new work has been done with Sodium Pentothal, and possibly this anaesthetic may be used more extensively when more definite knowledge is obtained.

3. Constant and expert supervision is needed over the administration of all drugs pre-partum.

4. The ideal analgesic agent for maternity work has not yet been found.

RÉSUMÉ

En obstétrique, depuis l'usage de l'anesthésie par la Reine Victoria, il est fait un usage courant de l'analgésie et de l'anesthésie. Au cours des vingt dernières années, on a mis au point des appareils permettant d'observer la respiration du foetus dans l'utérus et il est maintenant possible d'observer "de visu" les effets, sur la respiration du foetus, des substances anesthésiques et des drogues employées. Au cours de la première partie du siècle, on employait fréquemment la morphine et la scopolamine; aujourd'hui, à cause des effets contraires de ces drogues sur le foetus, on a réduit de beaucoup leur usage et on les a remplacées par le Demerol et le Phenergan. Comme substance anesthésiques, le cyclopropane, le protoxyde d'azote, le trichloréthylène et l'anesthésie régionale ont remplacé l'éther et le chloroforme. Pour les prématurés, toutes les drogues sont dangereuses. Pour combattre la dépression causée par les drogues sur le foetus, on ne devrait pas employer la nalline en même temps que l'anesthésie générale ou en même temps que des barbituriques.

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THE CARDIOVASCULAR REFLEXES

H. B. KEENLEYSIDE, M.D.*

THIS REVIEW is an attempt to discuss briefly some of the extremely diverse autonomic reflexes that affect the cardiovascular system. A large number of widely varied reflex phenomena are active in normal physiology, pathological physiology, and during surgical procedures.

The carotid sinus and aortic depressor reflexes constitute a mechanism of the utmost importance in the regulation of blood pressure (1). Pressoreceptors situated in the carotid sinus and aortic arch are tonically activated at any blood pressure above approximately 70 mm. Hg. (2, 3). The higher the arterial pressure the more rapidly do individual receptors discharge impulses, and the larger the number of receptors excited. Below about 70 mm. Hg, all pressoreceptor impulses cease. Thus when arterial pressure rises from normal to higher levels, pressoreceptors inhibit central vasomotor and cardio-accelerator tone. This results in vasodilatation, bradycardia, and reduction of blood pressure. As arterial pressure falls from normal to lower levels, pressoreceptor impulses decrease, and central vasomotor and cardio-accelerator tone rises. Thus vasoconstriction, tachycardia, and rise in blood pressure follow (1, 3, 4).

Early in haemorrhagic shock, the pressoreceptor reflexes maintain blood pressure by vasoconstriction; but when pressure levels fall to approximately 70 mm. Hg., the pressoreceptors are inactive, and unable to meet the emergency. However, the chemoreceptors of the carotid and aortic bodies become more active as blood pressure falls, and serve to forestall further reduction in pressure. The carotid and aortic body chemoreflexes, unlike the pressure reflexes, are an emergency mechanism for blood pressure maintenance. The chemoreceptors are maximally active under severe conditions, and require anoxia and hypercapnia as stimuli to activity. Although the pressoreceptor reflexes are easily depressed by anaesthetics, the chemoreceptors are not (3). Kenney and Neil (5) have recently shown that chemoreceptors discharge vigorously in haemorrhagic shock. Haemorrhage leads to reduced excitation of pressoreceptors and reflex vasoconstriction; and intense constriction of arterioles supplying the chemoreceptors also occurs. Blood flow through the chemoreceptors is thereby reduced, tissue oxygen tension falls and carbon dioxide accumulates. The chemoreceptors then become active even though the oxygen tension of blood leaving the lungs may be normal. The result is an increase in vasoconstriction and tachycardia, and a tendency to sustain the blood pressure.

The sino-aortic mechanism has been known for many years to be vitally important in the regulation of the circulation. Recently it has been discovered that there are reflex mechanisms of great complexity in the heart and lungs; these reflexes may ultimately be shown to play an important part in control of the circulation (6, 7, 8). Daly and Verney (9) described the left heart reflex. They observed a reflex slowing of heart rate when left ventricular pressure is

*Department of Anaesthesia, McGill University, Montreal.

raised. This reflex could supplement the aortic depressor reflex, and in fact there is histological evidence that stretch receptors in the aortic arch and left ventricle are part of the same system (6). The coronary chemoreflex (Bezold reflex) has aroused great interest (6, 8). A reflex fall of blood pressure and heart rate follows the injection of certain chemical substances into the coronary circulation. Substances which elicit the reflex are veratridine, serotonin, and adenosinetriphosphate, among others. Receptors for the reflex have not been found, but they must lie in the course of the coronary circulation. Serotonin is a substance released from disintegrating platelets, and adenosinetriphosphate may be liberated from damaged myocardium. Thus the possibility exists that circulatory collapse following coronary artery occlusion is partly reflex in origin.

A reflex fall of blood pressure and heart rate has been observed when the pressure in the pulmonary circulation is raised. This is the pulmonary depressor reflex (6). Its receptors lie in the pulmonary vascular bed. The purpose of this reflex may be to cause reflex systemic vasodilatation as a means of moving blood from the pulmonary to systemic circulation, when pressure rises to a dangerous height in the pulmonary vessels (8). Another depressor reflex is elicited by the injection of certain substances into the pulmonary circulation, or by inhalation of certain gases into the lower respiratory tract. This is the pulmonary depressor chemoreflex. It arises from receptors deep in the lung. Serotonin by injection and bromine by inhalation are two substances capable of evoking the response. The pulmonary chemoreflex may possibly become activated in pulmonary embolism. Experimentally, distension of the right or left pulmonary artery by a cuffed catheter produces no change in systemic blood pressure. Yet a profound fall of blood pressure and heart rate often accompanies multiple small pulmonary embolism. It has been suggested that serotonin, liberated from small pulmonary emboli, might activate the pulmonary chemoreflex and lead to circulatory collapse (6, 8).

These four depressor reflexes arising from the heart and lungs all have their afferent fibres in the vagi (6).

In the walls of the great veins and right atrium are vagal nerve endings which respond to changes in venous pressure. Much conflicting evidence concerning the physiological purpose of these nerve endings has accumulated (6, 7). Bainbridge (10) discovered that rapid infusions of large quantities of saline caused a reflex increase of heart rate. The stimulus for the reflex is presumed to be increased pressure in the great veins. Aviado and his colleagues (11) arrived at a completely opposite conclusion. These workers found that increasing the pressure in the isolated perfused right heart caused a reflex fall of blood pressure and heart rate. But it is not known what physiological purpose might be served by a depressor reflex caused by increased pressure in the right heart; the typical response in muscular exercise is cardiac acceleration and not slowing (8). Another reflex possibly arising from the right atrium was suggested by McDowall (12). He believed that reflex vasoconstriction following haemorrhage was due to a reduction of venous pressure in the great veins and right heart. He had insufficient evidence to support his view that the reflex arose from the venous side of the circulation. But Kenney and Neil (5) have shown that vasoconstriction

after haemorrhage occurs due to strong excitation of chemoreceptors. Thus the reflex originally described by McDowall as coming from the right heart may instead arise from the chemoreceptors (6, 13).

Reflexes causing changes in the circulation may be initiated by non-physiological stimuli during the course of surgical procedures. These reflexes exert deleterious effects on circulatory homeostasis. Such reactions usually arise when the plane of anaesthesia is light, since in this situation autonomic nerve pathways are active, at times even hyperactive. When reflexes of non-physiological origin occur during surgery, the homeostatic sino-aortic mechanism then functions in such a way as to oppose the untoward circulatory changes.

Stimulation of the carotid sinus during surgery of the neck may cause dangerous circulatory reactions (14). Pressure stimulation of a sensitive carotid sinus results in arterial hypotension and cardiac slowing or asystole. Hypersensitivity of the sinus reflex is most often found in elderly patients with arteriosclerosis or hypertension (15, 16). The reflex is also more active in hyperthyroid patients, and in those with infections of the neck (17). Lahey and Eversole (18) emphasize the necessity of differentiating the hypersensitive carotid sinus reflex from true cardiac arrest at operation. Asystole due to the sinus reflex is hardly ever fatal, and rarely persists longer than one minute after the initiating stimulus is removed. The sinus may be affected by external or internal pressure stimuli. External stimuli occur notably during thyroidectomy and incision of the cervical abscess in Ludwig's angina. The reflex may also follow pressure on the sinus by the anaesthetist's hand, or by shoulder braces used for support in the Trendelenburg position (19). Stephen, Nowill, and Martin (20) have noted the reflex after internal pressure stimuli. During the surgical repair of arteriovenous fistulae, hypotension and bradycardia may occur owing to increased pressure in the sino-aortic area. The injection of dye into the carotid artery for arteriograms may also initiate the reflex.

Burstein *et al.* (21) have studied the cardiocirculatory disturbances which may occur in intrathoracic surgery. Intercostal nerve stimulation due to rib periosteal scraping and rapid rib spreading may lead to auriculoventricular conduction defect, nodal rhythm, and arterial hypotension. Intrathoracic manipulations may cause direct vagus nerve stimulation and evoke sudden bradycardia and hypotension. The reactions following pleural incision are usually not severe. Arterial hypotension and bradycardia may result from manipulation of the pulmonary hilus, from direct mechanical pressure on the vagus nerve, or from traction on the oesophagus (21). Cardiac arrest has occurred following persistent traction on the hilus (22). Vagotomies should be performed low in the chest since the vagus nerve in this area is less sensitive to stimulation. The vagus at the level of the heart or higher in the chest is the most sensitive portion of the nerve (21). Manipulation of the pericardium, especially inflamed pericardium, results in severe reactions. They are more severe than those due to myocardial or intracardiac stimulation, and can be detected clinically as acute arrhythmias (23). Occasionally, severe reflex disturbances are observed at the time of ligation of the pulmonary artery and veins and clamping of the bronchus (24).

Mechanical irritation of the respiratory tract may cause bradycardia and hypotension by a vagal reflex (17, 25). The reflex may be set up by tracheal intubation, by inflation of an intratracheal cuff (26), or by mucus or gastric content inhaled into the larynx and trachea (27, 28). Reid and Brace (26) first described reflex bradycardia and cardiac arrhythmias following tracheal intubation. Since then there have been numerous reports of cardiac inhibition to the point of complete arrest due to intubation, and most ascribe the disturbances to a vagovagal reflex initiated in the respiratory tract (29, 30, 31). However, Burstein *et al.* (21) observed sinus tachycardia most frequently during intubation. Since then, King, Elder, Dripps *et al.* (32, 33), and Stephen, Martin, and Nowill (34) have also observed increase in heart rate and blood pressure following intubation.

Chemical irritation of the respiratory tract may also exert reflex effects on the heart (25). Johnstone (35) found that inhalation of cyclopropane or ether can produce bradycardia and serious cardiac arrhythmias, including cardiac arrest. This inhibition of the heart is probably caused by a vagal pulmocardiac reflex initiated by stimulation of vagal nerve endings in the air passages by the anaesthetic agent. Atropine will prevent serious reflex inhibition of the heart resulting from the inhalation of irritant anaesthetic agents. Johnstone (36, 37) has also shown that ether will abolish ventricular arrhythmias associated with cyclopropane anaesthesia, probably by stimulation of the vagal pulmocardiac reflex.

The exact nature of the celiac plexus reflex is controversial. Burstein and Rovenstine (38) described the reflex as due to increased tone of the splanchnic sympathetic nerves resulting from atropine administration, ether anaesthesia, and stimulation in the region of the celiac ganglion. They noticed that, during upper abdominal operations, the systolic blood pressure suddenly fell to a level almost as low as the diastolic pressure, though the diastolic pressure and pulse rate remained relatively normal. Burstein and Rovenstine believe that this marked narrowing of pulse pressure is probably due to diminished cardiac output. But the underlying mechanism for the reduction in cardiac output they did not explain. Reduced output of the heart could result from either reduced venous return or diminished force of cardiac contraction. Stimulation of the celiac plexus might lead to loss of tone of the great veins (39), and thus a reduction in venous return. Smith (40), however, believes that the reduced cardiac output is more likely due to decreased force of cardiac contraction. Experimentally, Burstein and Rovenstine (38) found that the celiac plexus reflex is intensified by atropine administration or vagal section, but alleviated by physostigmine. On the other hand, Smith (40) has recently observed that atropine will abolish a marked celiac plexus reflex. Smith therefore believes that the reduction in cardiac output is due to diminished force of cardiac contraction resulting from vagal inhibition of the heart.

Reflex circulatory changes during neurosurgery have been observed by Howland and Papper (41). The oculo-cardiac reflex is well known. The afferent limb of this reflex, the trigeminal nerve, is stimulated when a head rest presses on the eyes with the patient in the prone position. Bradycardia and hypotension result. An interesting series of disorders was observed during stimulation in various

parts of the cranium and its contents. The sites of stimulation were the galea, pericranium, dura, cortical veins, and temporal and frontal lobes. Stimulation was either mechanical or by means of the electrocautery. The changes consisted mostly of cardiac slowing and arrhythmias, and were likely due to vagal inhibition of the heart. On two occasions manipulation of the dura provoked cardiac arrest. They also noted bradycardia and apnoea following stimulation of the uncal gyrus of the temporal lobe with the electrocautery. Bergner (42) reports a case of reflex cardiac arrest following the application of pressure to the floor of the fourth ventricle.

Finally, it is of interest and importance to mention the "mass autonomic reflex" exhibited by subjects with high spinal cord injuries. Guttman and Whitteridge (43) observed that subjects with transection of the spinal cord at or above the fifth thoracic segment suffered mass autonomic reflex activity when distension of the bladder occurred. Manifestations of the diffuse reflex response are vasoconstriction, severe hypertension, bradycardia, excessive sweating, flushing of the face, headache, and blockage of the nasal air passages. Although such sympathetic overactivity most frequently results from distension of the bladder, it may also follow stimulation of almost any portion of the paralysed trunk, extremities, or viscera (44). Ciliberti, Goldfein, and Rovenstine (44) report numerous instances of severe hypertension during general anaesthesia in paraplegics.

Summary and Conclusion

The carotid sinus and aortic arch pressure reflexes are of supreme importance in the regulation of the circulation. They are tonically activated by normal physiological stimuli.

The physiological rôle of the carotid and aortic body chemoreflexes in hemorrhagic shock has been described.

Certain recently discovered reflexes, which may be important in normal physiology, have been briefly mentioned.

The physiological purpose of the venous pressure receptors is not yet established.

Deeper planes of anaesthesia depress the sino-aortic pressure reflex mechanism; and on this account fall of blood pressure due to surgical trauma is liable to occur. Light planes of anaesthesia do not depress this protective compensatory mechanism. However, during light planes of anaesthesia certain detrimental reflexes may be evoked by non-physiological stimuli associated with surgical procedure. The nature of a number of these untoward reflexes—carotid sinus, intrathoracic, and celiac plexus—has been reviewed. Reflex disturbances occurring during neurosurgery and in individuals with spinal cord injuries have also been mentioned.

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THE CAUSES AND TREATMENT OF COLLAPSE ON THE OPERATING TABLE*

J. G. ROBINSON, M.D.

STEDMAN defines collapse as a condition of extreme prostration similar to shock and due to the same causes, often with the addition of a great loss of fluid. To the anaesthetist, collapse generally signifies one of two things: first, primary cardiac failure, or, secondly, the more gradual deterioration of a patient's condition, which is conveniently described as shock and which, if severe and untreated, may lead to secondary cardiac failure. Thus secondary failure occurs because the patient has died whereas in primary failure the patient is still alive and will remain so for several minutes. Immediate vigorous treatment in this latter type should restore the heart action leaving the patient none the worse for his experience.

Anaesthesia, as it is generally practised by anaesthetists, surgeons, or physicians falls into several well-defined groups. The question of collapse will be considered as it occurs most commonly in each of the following groups.

A. Inhalation Anaesthesia

B. Conduction Anaesthesia
   (1) Spinal and Epidural Anaesthesia
   (2) Regional Block Anaesthesia
   (3) Local and Topical Anaesthesia

C. Intravenous and Rectal Anaesthesia

A. *Inhalation Anaesthesia*

In this type of anaesthesia, the most dramatic collapse occurs in the form of primary cardiac failure. Usually, in the early stage, the heart is either beating very feebly, in ventricular fibrillation, or quiescent.

Primary cardiac failure can occur in both normal and cardiac patients. This type of failure or arrest may arise from marked vagal stimulation such as stimulation of the trachea or be due to traction on the hilar regions of the lungs, or on splanchnic and mesenteric plexuses or the broad ligaments. It may result from anoxia produced by an over-dosage of the anaesthetic agent or by respiratory obstruction. Hypoxia and hypercapnia are important factors in potentiating the vagal effect on the pacemaker.

In 41 cases of primary cardiac failure, Turk and Glenn (4) list the causes as follows:

Sudden death, either from the condition which brings the patient to the operating room or from a complicating medical condition, may simulate primary cardiac arrest. Patients suffering from severe haemorrhage, trauma, toxaemia, or insulin shock form a part of this group. In any event, if, suddenly, no pulsations

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TABLE I

PRIMARY CAUSES OF CARDIAC ARREST

Cause	Number of Cases
Vagal Stimulation	
Direct	4
Reflex	1
Anoxia	
Haemorrhage	5
Obstructed airway	
Anaesthesia	2
Head injury	2
Coronary occlusion	1
Sedation	1
Drug idiosyncrasy	2
Direct endocardial stimulation	
Cardiac catheterization	2
Primary arrhythmia	
Acute adrenal insufficiency	
Uncertain	

NOTE: A few of these were preoperative and postoperative cases.

can be felt and no sounds can be heard over the praecordium, then the diagnosis of primary cardiac failure is made and no time must be lost in instituting artificial respiration and artificial circulation. A thoracotomy is performed and the heart massaged while oxygen from the anaesthetic machine is administered by manual control through an endotracheal catheter, if one is already in place, or by a face-piece. Fluids of any type may be administered intravenously under pressure. Intracardiac infusion of epinephrine (3–5 cc. of 1/10,000 solution) may initiate rhythm after massage has been started although ventricular fibrillation may be produced if the myocardium is still anoxic.

Ventricular fibrillation is often a product of light anaesthesia and frequently occurs early in induction or just before the moment of awakening. It may be closely associated with a sudden increase of epinephrine in the blood stream—either endogenous or exogenous. It may also follow another cardiac arrhythmia. The usual reasons for these arrhythmias are:

1. Severe oxygen lack.
2. Stimulation of autonomic reflexes with the vagal nerve as the efferent pathway.
3. Manipulation of the heart or great vessels.
4. Temporary circulatory obstruction.
5. Over-dosage of anaesthetic agent.
6. Excessive use of vasopressor drugs.
7. Manipulation of epinephrine producing tumours.

Treatment of ventricular fibrillation is, again, artificial respiration and circulation. Massage is instituted immediately to ensure good colour and tone of the heart muscle and then electrodes are placed on the heart to carry out the defibrillation technique. Two or three shocks, each lasting about one second,

are generally sufficient to bring the fibrillation to a stop and then massage and epinephrine treatment are instituted as for cardiac arrest. The usual defibrillating current required is 1–2 amperes of a 60 cycle, 110 volt current. It has been shown in the dog heart that currents of less than one ampere regularly produce fibrillation whereas the above-mentioned amperage stops fibrillation. Procaine hydrochloride injected into the right ventricle in amounts of 3–4 cc. of 1 per cent solution bathed over the epicardial surfaces may also prove helpful in initiating normal rhythm where fibrillation has been prolonged. Brock and Milstein state that in a series of thirty cases of fibrillation they felt that procaine in doses of 50–200 mg. intravenously was effective in abolishing nine episodes of fibrillation. All these steps in the treatment of ventricular fibrillation may have to be repeated several times before normal rhythm is regained and maintained. However, it may also happen that recovery to normal rhythm may occur while resuscitative measures are being prepared.

Operative shock or progressive hypotension is often a prelude to secondary cardiac failure unless the continuing deterioration is reversed. A major fall in blood pressure may simulate primary cardiac failure because of absent pulses, but cyanosis and pallor occur more slowly in the secondary form of failure. Often obvious haemorrhage or traction on viscera may be the clue to the cause of hypotension.

The usual causes of secondary failure can be enumerated as:

1. Loss of fluids through sweating, respiration, haemorrhage, extravasation.
2. Prolonged exposure to trauma.
3. Stimulation of traction reflexes.
4. Loss of heat from exposed viscera and cold inhaled vapours.
5. Over-dosage of anaesthetic, either relative or absolute.
6. Severe anoxia.
7. Sudden changes or extremes in positioning of patient.
8. Perforation of a viscus.

Blood replacement is the treatment for haemorrhage; but plasma and plasma substitutes can be utilized until blood is available. Vasopressors, moderate head down position, adequate oxygenation, and intravenous fluids are the accepted quartet for treatment of increasing hypotension, provided the noxious stimulus or other exciting factor has been removed, and blood loss has not been excessive. Levophed may be necessary during prolonged surgery or excessive trauma to maintain approximate normal blood pressure but should not be utilized as a substitute for blood replacement. Thus the avoidance of deeper anaesthesia than necessary, the prevention of loss of heat and moisture, the avoidance of rough handling of tissues, and the maintenance of a high alveolar oxygen level throughout the operation are the main points to be kept in mind, in order to prevent the onset of increasing hypotension. In a normal patient, surgical or anaesthetic shock need never be fatal, but in patients with marked myocardial degeneration, coronary stenosis, chronic debilitating disease with or without advanced age, death may occur rapidly. Thus the issue resolves itself into one of balance between cardiac effort and cardiac support in the matter of tissue oxygenation and replacement of blood or fluid loss. Jacoby, Flory *et al* (8) report a successful out-

come in a case in which 54 pints of blood were given in 2½ hours to offset the blood loss during surgery. They also gave one gram of calcium gluconate for each 2–3 pints of transfused blood to offset the deleterious effects of hypocalcemia.

The circulatory factors relevant to collapse in inhalation anaesthesia not only include accidents due to decreased blood pressure, but those due to increased blood pressure as well. Severe hypertension may be produced as follows:

1. By rapid asphyxia which occurs occasionally during an anaesthetic and is augmented at the start by initial struggling and breath-holding.
2. A slow increase in blood pressure caused by poor technique which prevents adequate removal of carbon dioxide.
3. Increased epinephrine in the circulation due to fear, excitement, and struggling.
4. Overhandling of a toxic thyroid gland or adrenal tumours.

These four factors may produce acute cardiac dilatation or rupture of a blood vessel. Cardiac dilatation may restrict coronary flow, causing the heart to stop, while rupture of a vessel may cause immediate death. Obviously the treatment of these two conditions lies only in their prevention with emphasis on smooth, quiet induction and the maintenance of a free airway with attention to adequate removal of accumulating carbon dioxide.

Respiratory failure under general anaesthesia can be partial or complete but in either case, if allowed to persist long enough, will cause vasomotor collapse through prolonged anoxia. Respiratory failure can be considered as either central or peripheral respiratory failure.

Central respiratory failure is generally caused by:

1. Overdose of pre-anaesthetic sedative.
2. Overdose of the anaesthetic agent.
3. Increase of intracranial pressure.

One of the above factors may lead to serious trouble but a combination of two or more of these factors may well be disastrous.

When pre-anaesthetic medication has lowered the respiratory rate, the increased level of carbon dioxide in the blood will raise the normal respiratory threshold. Hyperpnoea of induction as in vinethene or ether induction will lower the carbon dioxide level and respirations may cease. Anaesthesia in a closed circuit will maintain blood carbon dioxide at more normal levels so that apnoea can be avoided. However, under prolonged closed anaesthesia elevated carbon dioxide levels may drop seriously at the conclusion of the anaesthetic with resultant hypotension and/or respiratory failure as is occasionally seen in so-called "cyclopropane shock." This somewhat rapid drop in blood pressure can be avoided by gradual elimination of the opiate, anaesthetic, and carbon dioxide towards the end of the operation by conversion to a high flow of nitrous oxide and oxygen in a semi-closed system.

Children, because of inadequate pre-anaesthetic sedation, often show hyperpnoea throughout induction with resultant blow-off of carbon dioxide and precipitate anaesthesia which further depresses respiration. Apnoea and cyanosis occur, and these are followed by frantic resuscitative measures on the part of

the anaesthetist. This situation can be avoided through adequate preoperative sedation and therefore quiet induction.

Any intracerebral or medullary pathology may elevate the respiratory threshold so that further elevation by opiates before operation or administration of the anaesthetic may completely paralyse respiration.

Peripheral respiratory interference occurs in such conditions as:

1. Breath-holding—due to direct irritating vapours.
2. Relaxation of jaw allowing tongue to occlude respiratory tract.
3. Laryngeal spasm—due to direct irritation of vocal cords or from abdominal reflex origin.

These conditions may lead to apoplexy or acute cardiac dilatation or to the effects resulting from prolonged hypoxia.

4. Aspiration of debris during induction or anaesthesia, which can lead to partial or total collapse of lung with subsequent hypoxia, collapse, and death.

The usual causes for this accident are:

1. Vomitus—in accident cases, obstetrical patients, those with intestinal obstruction, or poorly prepared patients.
2. Excess mucus.
3. Pus from pharyngeal or peritonsillar abscesses.
4. Bronchiectasis or lung abscess.
5. Haemorrhage from surgery or anaesthetic trauma to nose, throat, and mouth.
6. Broken teeth or suction tips.
7. Sponges or pharyngeal packs.

The use of lavage where a full stomach is suspected is a safety precaution that cannot be overemphasized. Often, however, particles of undigested food will still remain in the stomach so that smooth induction, the presence of adequate suction, an operating table that can provide immediate Trendelenburg, and an orotracheal cuffed catheter are all indispensable implements in the handling of the suspect "full stomach."

However, the above-mentioned accidents still occur and the restoration of pulmonary function is the prime concern in the institution of resuscitation. If no foreign obstruction is present in the pharynx or in the glottic region then intubation, oxygenation, and suction are the prime points in immediate resuscitation. Bronchoscopy should be carried out to complete the removal of vomitus, blood clots, or foreign material from the trachea or bronchi. Anoxia during the bronchoscopy should be studiously avoided, since the patient's hypoxic state can easily be further aggravated during the insertion and manipulation of the bronchoscope.

Other pulmonary conditions which, if left unattended, can lead to hypoxia and collapse are:

1. Internal or external paradoxical respiration.
2. Bronchopleural or tracheal fistula.
3. Bilateral pneumothorax.
4. Acute emphysema.
5. Pleural effusion.
6. Pulmonary oedema.

External paradoxical respiration can be treated by providing an unobstructed

orotracheal airway. Internal paradoxical respiration occurs in the open chest and is handled by controlled respiration. Bronchopleural or tracheal fistula are generally accidentally produced and allow escape of vital oxygen and anaesthetic agent. High flow oxygen, intravenous anaesthesia, if necessary, and repair of the damage are the necessary points in the treatment. Bilateral pneumothorax may occur in surgery of the oesophagus, trachea, or the great vessels and controlled respiration via an endotracheal catheter is the bulwark against collapse. Acute emphysema following ball-valve obstruction of a bronchus may require bronchoscopy or bronchial exploration to relieve the obstruction. Rupture of a lung cyst following positive pressure by the anaesthetist may cause pulmonary collapse so that immediate reparative surgery may be required before this complication progresses.

Undiagnosed pleural effusion may be present in cardiac patients or patients presenting with sub-phrenic or pancreatic abscesses. Respiratory embarrassment may become quite marked during or at the end of the operative procedure.

Pulmonary oedema is a serious complication which, fortunately, develops rarely. It is characterized by gentle wheezing and the appearance of pinkish fluid in the tracheobronchial tree. The usual causes centre around damage to pulmonary capillaries by

1. Prolonged shock.
2. High pulmonary intravascular pressure from cardiac dilatation or cardiac tamponade.
3. Excessive negative intratracheal pressure, i.e. respiratory obstruction.
4. Direct injury as in an anaesthetic explosion.
5. Prolonged extreme head down position.
6. Overhydration.
7. Anaphylactic reaction.
8. Congestive heart failure.

Treatment consists of tracheobronchial toilet and the application of slight positive pressure during expiration. Intravenous infusion should be stopped or slowed and the table tilted to a 30° Fowler's position. Aminophylline (7½ gr. i.v.) should be given slowly and may be repeated if necessary. If the oedema is considered to be cardiac in origin, then digitalization should be carried out with 0.5–1 mg. of digoxin which may be repeated if necessary. Cortisone therapy may be required if oedema was due to explosion or anaphylactic reaction. Phlebotomy and administration of hypertonic glucose may prove useful. If blood was administered during the surgical procedure, then a recheck of the blood grouping should be carried out.

There are many other accidents that can precipitate collapse under general anaesthesia, a few of which deserve separate mention.

(1) *Tracheal Collapse.* This may occur following removal of a large thyroid gland with resultant respiratory obstruction. Intubation either should be done at the start of the thyroid operation or at least be anticipated throughout the procedure.

(2) *Convulsion.* Deep ether convulsions occur chiefly in children but may occur in adults as well. They may also occur in light anaesthesia. Controversy

exists as to whether deficiency or excess of carbon dioxide is the chief cause although quite often a febrile condition co-exists. Regardless of the cause, treatment consists of removal of the anaesthetic, administration of oxygen, and the intravenous administration of Pentothal.

(3) *Venous or Arterial Air Embolism.* These conditions prevent adequate cardiac emptying and filling with marked reduction in cardiac output and may lead to shock or immediate death. Massive amounts of air may be sucked or forced into the circulatory system after intravenous fluids are exhausted. Air embolism can also occur in neurosurgery and pelvic surgery and may lead to sudden cardiac failure. The patient should be placed in the supine position, turned on the left side, and given oxygen. If a normal heart beat is not restored, a needle puncture of the right ventricle should be performed in an attempt to aspirate the air, and if this fails, the air should be aspirated through a thoracotomy incision and massage instituted if necessary.

(4) *Ether Accidents.* (*a*) Liquid ether in lungs may occur when the ether vaporizer is wrongly connected. Violent pulmonary congestion, oedema, and death may ensue. This apparatus should always be tested before use. The use of a Flagg can will allow ether to be spilled into the lungs if it is tipped. These cans should always be strapped to the table after the patient is positioned. (*b*) Boiling ether is another factor. Some vaporizers have a heating arrangement to vaporize ether more efficiently, especially when adults are being anaesthetized with pharyngeal insufflation. When anaesthesia deepens the hook should be removed from the mouth—it is not sufficient to turn off the air current.

(5) *Miscellaneous.* Explosions or fire, metabolic emergencies, mechanical or human errors in anaesthetic techniques are mentioned for the sake of completeness and should not be minimized.

B. *Conduction Anaesthesia*

Toxic reactions to the drugs employed in the use of spinal and epidural anaesthesia, regional and local anaesthesia are due either to: sensitivity to the drug or to a high blood level of the drug. Sensitivity to the drug provides less than 2 per cent of the reactions seen and the wheals, itching, hypotension, and asthmatic breathing are treated symptomatically.

High blood levels of the agent used account for more than 98 per cent of the reactions seen and these reactions are more severe in nature. High levels arise mainly from inadvertent intravascular injection or injection into a highly vascular area. The use of hyaluronidase spreads the absorptive area and may give excessively high blood levels. Errors in dosage and faulty detoxification mechanisms in the patient also raise the blood levels.

The signs and symptoms are central in origin and are listed as follows:

1. Mental—drowsiness, dullness and coma.
2. Cardiovascular—Tachycardia progressing to bradycardia and hypotension, possible cardiac arrest.
3. Respiratory—Depressed shallow respirations, dyspnoea or bouts of apnoea.
4. Neurological—Minimal twitching or generalized convulsions.

These signs may be immediate or delayed up to thirty minutes. Thus the anaesthetist must be prepared to administer oxygen, vasopressors, atropine, barbiturates, and even perform cardiac massage if necessary for at least one half hour after the block has been given. It cannot be overstressed that immediate access to operating room facilities is of prime importance whenever block procedures are contemplated because of the possibility of toxic reactions to the drugs employed.

In the first half hour of spinal anaesthesia the most dramatic form of collapse is that of a combined respiratory and circulatory depression. The tone of the entire vascular bed is maintained both by the action of the sympathetic innervation and by the tone of the voluntary muscles. Thus the combined effect of sympathetic paralysis, hypoxia due to respiratory depression, and loss of muscular tone is to produce circulatory and respiratory depression which varies directly as the height of anaesthesia progresses. This condition may go on to severe collapse and cardiac slowing by paralysis of the cardiac accelerator nerves.

Treatment consists of:

1. Intravenous infusions of saline or glucose with vasopressor administration through the rubber tubing.
2. Oxygen administration by mask with positive pressure.
3. Horizontal positioning of patient since head up or head down positions may further advance the hypobaric or hyperbaric spread of the anaesthetic solution or conversely with result in imperfect anaesthesia so that added agents will be required after normal tension has been restored.

With profound vasomotor and respiratory collapse cardiac standstill may occur. Treatment consists of:

1. Instant recognition.
2. Scultetus position to provide blood for the vital centres.
3. Cardiac massage.
4. Oxygen mask under positive pressure—later by endotracheal tube.

Respiratory and circulatory collapse under spinal anaesthesia can occur individually or combined and it must be stressed that either can set up a chain of events that will bring about the other.

Hypertension may occur with resultant cardiac dilatation, rupture of a cerebral vessel, or cardiac arrest. The cause may be asphyxia, adrenalin excess, too much sympathetic stimulation prophylactically or from the toxic effect of the vasoconstrictor agent used. Treatment of hypertension consists of assisted respirations in a closed circle filtre, aminophylline 3¾ gr. i.v., 50 mg. doses of Pentothal, or if available a nitroglycerin tablet 1/100 gr. placed under the tongue.

Epidural anaesthesia can produce hypotension to about the same degree as spinal anaesthesia although respiratory paralysis is not as marked. Inadvertent puncture of the outer meninges and flooding of the canal with the agent used may produce spinal anaesthesia past the foramen magnum with profound vasomotor and respiratory collapse. This same complication may also occur in stellate ganglion block, thoracic or lumbar paravertebral block, caudal block, and even in a brachial block. The usual precautions of aspiration, followed by the injection of a small amount of the drug and several minutes' scrutiny for any untoward

reactions, should always be taken. In the event of a misadventure, treatment is the same as that already outlined for spinal circulorespiratory collapse.

The use of topical anaesthetics such as cocaine, butyn, pontocaine, nupercaine as employed in rhinology, laryngology, ophthalmology, and genito-urinary procedures, is fraught with the same dangers as those encountered in regional or local block procedures. With regard to the use of barbiturates before operation to offset these toxic phenomena, Moore (2) is of the opinion that barbiturates given before operation are useful only to allay apprehension. Moore states that to prevent a toxic reaction, barbiturates would have to be given in anaesthetic levels. Stimulation of the central nervous system, progressing to eventual depression and paralysis of respiratory and vasomotor centres, is the usual sequence of signs of drug toxicity. The treatment for such reactions consists of oxygen, suction, vasopressors, and small doses of intravenous barbiturates to stop convulsions.

C. *Rectal and Intravenous Anaesthesia*

The relative or absolute over-dosage of anaesthetic agents in rectal or intravenous anaesthesia constitutes the main cause of mishaps in these forms of anaesthesia. Barbiturates by rectum or intravenously can cause profound respiratory and circulatory collapse and the treatment is supportive. Other forms of rectal anaesthesia produce their ill effects through respiratory depression and resultant anoxia. Intravenous procaine carries with it the dangers of toxic manifestations and in higher doses may lead to cardio-respiratory collapse through its action on central and autonomic nervous systems.

Summary

This article has been designed to review some of the more important forms of collapse on the operating table and in most instances the cause and treatment of each form of collapse have been outlined briefly. Collapse under inhalation and conduction anaesthesia has received the bulk of attention in this review since rectal anaesthesia is not a common procedure and intravenous anaesthesia in all its newer forms was considered to be beyond the original scope of this article. Individual surgical techniques which in themselves might prelude collapse were purposely omitted and instead an effort was made to keep the subject within the anaesthetist's domain.

Résumé

Nous avons passé en revue les différentes formes de collapses pouvant survenir sur la table d'opération ainsi que leurs causes et leurs traitements; nous avons envisagé le problème selon qu'ils surviennent au cours de (*a*) l'anesthésie par inhalation; (*b*) l'anesthésie loco-régionale; (*c*) l'anesthésie rectale et intraveineuse.

Sous anesthésie générale, nous avons l'impression que la défaillance cardiaque primitive survient fréquemment à la suite d'un effet vagal sur le centre d'automatisme résultant d'une stimulation vagale soit directe soit réflexe. L'anoxie et l'hypercapnée sont des facteurs importants quand survient la défaillance cardia-

que primitive. Une complication médicale ou chirurgicale peut également produire le même tableau clinique d'arrêt cardiaque. Les premiers gestes de la resuscitation sont les suivants: le diagnostic précoce, la thoracotomie, le massage cardiaque, l'oxygène sous pression et l'adrénaline intracardiaque si nécessaire. La fibrillation ventriculaire survient sous anesthésie légère, en présence de quantité excessive d'adrénaline, en déficience d'oxygène ou, encore, à partir d'une autre arythmie; on convertit cette fibrillation en arrêt cardiaque par la défibrillation électrique puis on institue le traitement de l'arrêt cardiaque. Certains auteurs ont employé avec succès de la procaine à 1 pour cent soit en injections intracardiaques (3–4 cc.) soit en injections intraveineuses (50–200 mgm.) dans le but de restituer un rythme normal lorsque la fibrillation s'est prolongée.

La défaillance cardiaque secondaire est une autre sorte de collapse; elle peut survenir au cours de l'anesthésie générale et elle est le résultat d'une aggravation progressive de l'état du malade. Cette sorte de collapse peut résulter soit d'une perte de liquides, d'un traumatisme prolongé, de réflexes de traction, soit d'un surdosage anesthésique surtout si ces facteurs ne sont pas contrôlés souvent. Dans ces cas, il ne faut jamais employer de vasopresseurs à la place de sang ou de plasma. Au cours de cette sorte d'anesthésie, il peut survenir, à l'occasion, une hypertension marquée. Une élimination inadéquate du gaz carbonique, de l'appréhension et de l'agitation, des manipulations exagérées d'une thyroïde toxique ou des surrénales peuvent conduire à cette situation et causer une dilatation du cœur ou la rapture d'un vaisseau.

Sous anesthésie générale, la défaillance partielle ou complète de la respiration, pour peu qu'elle se prolonge, peut également entraîner un collapse circulatoire par anoxie prolongée. La défaillance respiratoire centrale peut survenir à la suite de surdosage de la prémédication ou de l'agent anesthésique ou encore d'une augmentation de la pression intracranienne. La défaillance respiratoire périphérique est plus fréquente; elle comprend plusieurs formes d'obstruction des voies respiratoires, mécaniques, physiologiques ou pathologiques, obstructions qui peuvent réduire ou empêcher l'oxygénation.

Avec l'anesthésie loco-régionale, on observe occasionnellement des réactions aux drogues employées. Ces réactions apparaissent dans les cas de sensibilité particulière aux médicaments ou lorsqu'il y a un taux élevé du médicament dans le sang. Moins de 2 pour cent des réactions anormales sont dues à une sensibilité spéciale et elles peuvent être traitées symptomatiquement, tandis que 98 pour cent de ces réactions sont provoquées par un surdosage et elles peuvent être sérieuses. On peut observer des manifestations psychiques cardiovasculaires, respiratoires et neurologiques dont le traitement requiert de l'oxygène, des vasopresseurs, de l'atropine, des barbituriques et, dans un nombre de cas plutôt restreint, le massage cardiaque peut devenir nécessaire. En conséquence, quand on projette de faire un bloc, il faut toujours avoir à sa portée les facilités dont on dispose dans les salles d'opérations.

Au cours de la rachianesthésie, la complication la plus dramatique est le collapse simultané de la circulation et de la respiration. La bradycardie peut aussi devenir un facteur important. Le traitement doit tendre à lutter contre l'hypotension et la manque d'oxygène. L'hypertension et ses complications peu-

vent avoir pour causes: le manque d'oxygène, l'excès d'adrénaline, endogène ou exogène, ou encore, une stimulation sympathique prophylactique trop intense. Les blocs épiduraux également peuvent produire de l'hypotension; toutefois les collapses plus sérieux qui surviennent sont dus à l'inondation imprévue de l'espace sousarachnoïdien par l'agent anesthésique. Cette éventualité peut également se produire à l'occasion d'un blocage du ganglion étoilé, d'un blocage paravertébral thoracique ou lombaire, d'un blocage caudal ou brachial. Pour prévenir cette inondation subarachnoïdienne, les détails importants sont: l'aspiration, l'injection de petites quantités et des périodes d'attente.

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RESPIRATORY OBSTRUCTION DUE TO BLOOD CLOT FROM BLEEDING BRONCHIECTASIS:

A Case Report

GORDON M. WYANT, M.D., F.F.A.R.C.S.*

It has long been recognized that flooding of the opposite lung during thoracotomy is a grave danger in all cases where there is fluid or semi-solid material in the affected side of the lung. This danger is particularly great in wet bronchiectasis, lung abscess, and haemoptysis. The following case is reported because it once again emphasizes this grave danger.

CASE REPORT

History. Patient: Mrs. G. F., a white female, aged 25 years. She was admitted to University Hospital on December 24, 1955, because of haemoptysis. She gave a history of whooping cough at the age of 6 years and persistent morning cough with greenish sputum up to 2 or 3 tablespoonsful in amount each morning. Six years previously she had coughed up a little blood and one year thereafter she had had another bout of haemoptysis. She was married in 1952 and had no further complaints until following the birth of her second child in November 1955. Two weeks post-partum she coughed up blood for three nights running, about one cupful each time. She was admitted to her local hospital on December 21, 1955, where she had two more episodes of bleeding with as much as half a kidney basin of blood each time. Because of this, she was transferred to University Hospital by air ambulance.

Examination. The patient was a rather thin woman weighing 105 pounds, in physical state III on account of the history of repeated haemoptysis and diminished movement in the left thorax as well as slight dullness over both bases which was more marked on the left side. There were moist rales in both bases. Otherwise her physical condition was not remarkable. Her haemoglobin on admission to hospital was 10.9 gm. and P.C.V. 32 per cent. Although only one unit of blood was given, her preoperative haemoglobin had risen 5 days later to 12.3 gm. and P.C.V. to 36 per cent. X-ray on the day of admission showed both costophrenic angles well defined and clear lung fields. On December 29, however, X-ray of her chest showed a rather hazy, fluffy type of increased density over both bases, compatible with the aspiration of blood. She had two further episodes of haemoptysis of a cupful each on December 26 and again on December 29, and it was decided that operation should be done the following morning.

Anaesthetic management. The patient was premedicated with meperidine (Demerol®) 50 mg., scopolamine hydrobromide 0.4 mg., given intramuscularly at 6:50 A.M. She arrived in the operating room at 7:30 A.M. well sedated but not depressed. Her blood pressure was 110/80, pulse 88, and respiration 20. Anaesthesia was induced with dimethyltubocurarine (Metubine®) 5 mg. followed by sodium thiopental (Pentothal®) 2.5 per cent, 0.375 gm. In order to avoid all bucking which might stir up new bleeding, topical anaesthesia of trachea and larynx was carried out with tetracaine (Pontocaine®) 1 per cent, 20 mg. and a No. 9 cuffed endotracheal tube lubricated with jelly cyclomethycaine (Surfacaine®) 0.75 per cent was inserted with ease. There was no bucking upon intubation. Anaesthesia was maintained in a light plane with cyclopropane

*From the Department of Anaesthesia, University of Saskatchewan College of Medicine and University Hospital, Saskatoon, Saskatchewan.

and ether, and controlled respiration was instituted immediately after induction. The patient was placed in the right lateral position, and operation was started 20 minutes after induction of anaesthesia. There was at that time no evidence of moist sounds in the tracheo-bronchial tree. The pleura was opened approximately 10 minutes after skin incision and 10 minutes later, during exploration, some few moist sounds were noted and repeated tracheo-bronchial toilet was carried out. One hour and fifteen minutes after induction of anaesthesia, tracheo-bronchial toilet revealed the presence of a little blood, but this did not seem to be excessive and was aspirated. However, 10 minutes later sudden almost complete respiratory obstruction occurred which lasted for close to 10 minutes during which time the pulse rate fell to about 60 and there was increasing cyanosis which eventually merged into a degree of pallor. Patency of the endotracheal tube having been ascertained and mechanical defects in the machine having been rapidly excluded as possible causes, repeated tracheo-bronchial toilet was vigorously continued and eventually it became possible to re-inflate the exposed upper left lobe; this was followed by some improvement in the patient's condition. It was suspected at that time that ventilation of the right lung was probably poor or absent, judging by the resistance in the breathing bag. By that time the pulse rate had risen to between 130 and 140; the blood pressure had remained pretty well stable throughout the episode of difficulty and thereafter. Tracheo-bronchial toilet was continued repeatedly while arrangements were made for bronchoscopy, but before they were completed the suction catheter was made to enter the right main stem bronchus and on its withdrawal a long clot, measuring approximately 5 inches, was removed. This clot did not seem to be new in its entirety but there was suspicion of some organization of at least part of it, with fresh clot surrounding it. Soon after removal of the clot, the patient's colour improved markedly and within half an hour her pulse rate had subsided to somewhere in the neighbourhood of 110, where it remained throughout. The lobe was removed approximately one hour and forty-five minutes after incision and two further tracheo-bronchial toilets yielded no further blood. The remainder of the operation proceeded uneventfully, and at the end of the operation a total of 1200 ml. of blood, 750 ml. of 5 per cent Dextrose in water, and 150 ml. of normal saline had been given; the patient seemed to be in good condition, well oxygenated, with good blood pressure and pulse. Protective reflexes were present.

At the conclusion of the operation the patient was turned on her back having previously been given atropine sulphate, 0.4 mg. intravenously. Succinylcholine 30 mg. was administered and a 7 mm. bronchoscope was passed with ease. The trachea was entirely free of blood and none was found in the left main stem bronchus. However, a small clot, partially filling the lumen of the right main stem bronchus, was seen and was easily removed by suction. Thereafter the right bronchus and its subdivisions were found to be clean, as far as they could be visualized. The patient was then transferred to the recovery room, and placed in a high humidity oxygen tent.

Postoperative course. The postoperative course was quite smooth on breathing exercises and postural drainage. Postoperative films showed some degree of atelectasis and density over the left base but nothing was seen on the right side. No fluid levels were demonstrated at any time. The pathological diagnosis was cylindrical bronchiectasis and unresolved bronchopneumonia with organization.

Discussion

It was obvious to us before induction of anaesthesia that some form of safeguard should be established if possible to protect the unaffected right lung. Unfortunately, some difficulty had been experienced in obtaining all the equipment which had been ordered many months before for the Department, and neither Carlens tubes nor endobronchial blockers were available. The only right Bonica endobronchial tube available had been cut up by an orderly after

previous use in a similar case, and was found not to be serviceable on the day of operation. Consequently, very much against better judgment, mere endotracheal intubation was done, with reliance placed entirely on frequent aspirations and the fact that the surgeon had promised to co-operate by exerting pressure on the hilar structures in order to prevent undue escape of blood into the remainder of the tracheo-bronchial tree. Unfortunately, the hilum was so greatly involved in old inflammatory reaction that he was unable to give much assistance. Nevertheless, no difficulties were encountered until it became necessary to roll the lobe over and approach the hilum from behind in the course of dissection, and it was then that some blood must have escaped from the left side into the right main stem bronchus. From the appearance of the clot, it was evident that some old blood had become organized in the right bronchus but did not entirely obstruct it and that relatively little additional blood was needed to form a complete obstruction.

This case once again emphasizes the absolute need of managing all wet lungs from whatever cause by some means other than simple endotracheal anaesthesia; it would appear now that it would have been wise in the absence of suitable equipment to use a full-length endotracheal tube and cut a very long bevel which would hold the tube in the right bronchus without obstructing the eparterial bronchus, as suggested by Hall. The Overholt position would have been of advantage also.

Summary

A case of complete respiratory obstruction due to "flooding" from bleeding bronchiectasis is described. A clot, approximately 5 inches long, which had completely obstructed the right main stem bronchus was removed by suction before the patient had come to serious harm. This case is reported in order to emphasize the need for protecting the opposite lung in all "wet" cases, whether due to bronchiectasis, lung abscess, or bleeding, by such devices as a double lumen catheter, endobronchial blockers, or unilateral bronchial intubation.

A NEW FRAME FOR THE POSITIONING OF PATIENTS FOR SURGERY OF THE BACK

IAIN M. MACKAY, M.D., F.A.C.A.*

THE POSITIONING of patients for surgery of the back, especially of the spinal column, is a problem of equal importance to both surgeon and anaesthetist. While the primary consideration of each is different it is obvious that for the ultimate benefit of the patient their viewpoints must coincide closely.

There can be no doubt that the best possible operating conditions contribute to the success of any operation and to the recovery of the patient. However, it is most important that these conditions should not be achieved by imposing upon the patient other conditions unnecessarily disturbing to his physiological integrity.

Although for some operative procedures on the spinal column and related structures the patient may be placed in the lateral position or occasionally in the sitting position, the use of the prone position is favoured by most surgeons. For optimal operating conditions, particularly for surgery involving the lumbosacral vertebrae, various degrees of flexion or of extension of the back must be achieved. It is also desirable to be able to alter the position if the procedure should require it for best results. Unfortunately, the prone position of the anaesthetized patient, particularly the obese individual, on the ordinary operating table tends to diminish respiratory excursion or even to cause actual respiratory obstruction because pressure of the abdomen prevents proper functioning of the diaphragm. Normally responsible for roughly 60 per cent of the lungs' expansion, the diaphragm is even more important in the prone anaesthetized patient and interference with its excursion leads to both respiratory and cardiovascular complications.

Inadequate respiration leads inevitably to anoxia and carbon dioxide retention, and, because of an increased blood pressure, may cause excessive blood loss at the operative site. More important, perhaps, in contributing to blood loss and congestion in the surgical field, is the compression of the great veins of the abdomen, resulting in back pressure and in diminished venous return to the heart.

During the past year we have used in a number of cases a new positioning frame which has helped to solve our primary problems (Fig. 1). It consists essentially of two curved, heavily padded longitudinally placed metal bars, hinged at their mid point so that the height of the frame's apex and the degree of curve may be readily adjusted. These curved bolster bars are laterally adjustable by manual positioning and, once set, may be locked. The degree of curve is controlled by a longitudinally and centrally located screw which is operated from the foot of the table by a three-foot handle. This frame, if properly used, allows complete freedom of the abdomen. It allows a great range of positioning of the spinal column varying from extreme flexion to hyperextension. It is sturdy, cheap, and simple and has achieved some degree of popularity with the ortho-

*Department of Anaesthesia, University of Toronto, and Toronto General Hospital.
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FIGURE 1. Frame in position on operating table.

paedic surgeons and neurosurgeons in the Toronto General Hospital. However, like any other piece of apparatus, it can be misused. Careful positioning of the patient is still essential.

If the bolster bars are placed too close together they may themselves obstruct the abdomen. If they are placed too far apart the patient may fall between them. They are best adjusted to support the patient at the anterior superior spines of the ilium and in the mid-clavicular line in the thoracic region. The bolster bars are thus often in the best position when not parallel, but somewhat closer together at the upper end than at the lower.

The patient should be so placed that the operative site is at the highest point of the curve. If the position is any lower than this, maximal flexion cannot be

obtained. This is particularly true if the knees are allowed to rest on the table, in which case the pelvis remains tilted posteriorly. The patient's arms should be extended above the head and should rest on the head of the table (Fig. 2).

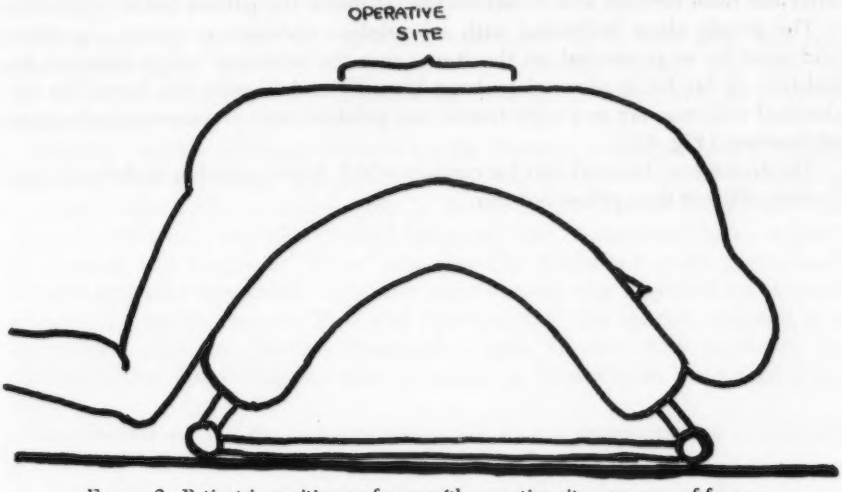

FIGURE 2. Patient in position on frame with operative site over apex of frame.

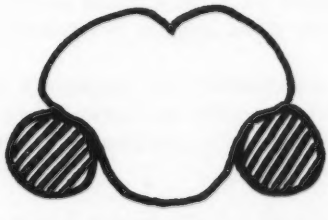

FIGURE 3. Cross-section of obese patient resting on bolsters of frame.

Following lumbosacral fusion it is often desirable to transfer the patient to a Stryker Frame while he is still in the prone position. The bulk of our frame makes it impossible to fit it between the sections of a Stryker Frame. However, a canvas lifter has been devised which may be placed under the patient before operation.

The grossly obese individual with a pendulous abdomen is always a problem and must be so positioned on the frame that the abdomen hangs between the bolsters. If his fat is allowed to hang laterally and outside the frame his abdominal wall may act as a tight binder and produce both respiratory and venous obstruction (Fig. 3).

The frame may be used also for cases in which hyperextension is desirable and is more efficient than pillow bolsters.

NEWS LETTER

Newfoundland

Growth of the specialty of Anaesthesia continues. Up to 1954 there was only one Newfoundland doctor in the Canadian Anaesthetists' Society, now there are ten who are members or in the process of joining. Of these, five are Certified Anaesthetists.

Regular monthly meetings are held for the Province in the St. John's General Hospital. During the past year anaesthetists were active at medical meetings of a general nature. Dr. A. Levitz spoke to the St. John's Clinical Society on "The Use of Intravenous and Intra-Arterial Infusions" and "Anaesthesia as an Adjunct to Therapy and Diagnosis." On a later date Dr. Stentaford spoke to the same Society in a joint symposium with two other doctors, one a general practitioner and one a paediatrician on "Neonatal Emergencies." On another occasion at a Refresher Course for General Practitioners from all over Newfoundland, Dr. Stentaford and Dr. Henderson gave a lecture on "Anaesthesia in General Practice."

Drs. Stentaford, Levitz, and Henderson will start a group practice in the near future, to be known as the Newfoundland Anaesthesia Associates.

Quebec

Dr. Ronald Millar, formerly of Edinburgh and Philadelphia, has joined the Anaesthetic Staff of the Montreal Neurological Hospital.

Dr. Léon Longtin, of the Hôtel-Dieu, Montreal, gave a demonstration on bronchial intubation at the Montifiore Hospital, New York, in March.

Ontario

Recent visitors to the University of Toronto, Department of Anaesthesia were:

Dr. Otto Mollestad, Director of Anaesthesia, Rikshospitolet, Oslo, Norway, who has toured various American centres, under the auspices of the American College of Surgeons.

Dr. Edward G. Hudson, Senior Anaesthetist of the Singapore Government, and Lecturer in Anaesthetics, University of Malaya, under the auspices of the World Health Association.

Dr. C. Ritsema van Eck, Groningen, The Netherlands, visiting Montreal and Toronto, sponsored by the American College of Surgeons.

Dr. David Little, of Hartford, Connecticut.

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The Executive of the Section of Anaesthesia, Academy of Medicine of Toronto, for 1956–57 is composed of Dr. Foster Smith, Chairman, and Dr. G. D. M. Boddington, Secretary-Treasurer.

Drs. Michael Campbell and Thomas Hanley, now finishing their postgraduate course in Anaesthesia at the University of Toronto, are leaving in July for Great Britain for further studies in Edinburgh and Oxford on R. S. McLaughlin Fellowships.

°

Dr. Ralph Hargrave, recently retired as the Anaesthetist-in-Chief, Toronto Western Hospital, is now Director of Anaesthesia in the new Scarborough General Hospital.

°

It is with regret that we report the sudden death of Dr. Maurice Lunau of Oakville, on April 10, 1956.

Alberta

It is with regret that we report the death of Dr. Ruth Johnstone Harvey, of Olds, Alberta, in December, 1955.

°

A plaque is to be erected in the Medical College of the University of Alberta, in recognition of the early work of Dr. Irving Bell and Dr. Gelfan with di-vinyl ether.

General

The Budget announcement of the Hon. Mr. Harris, that expenses for two recognized conventions in Canada are deductible for Income Tax purposes, is again drawn to your attention. The annual Canadian Anaesthetists' Society Meeting is a recognized convention and acceptable for this purpose.

°

Executives of the Divisions of the Canadian Anaesthetists' Society for 1956–57 reported to date are:

Newfoundland	*Chairman*	Dr. C. D. Kean
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Prince Edward Island	*Chairman*	Dr. L. Prowse
Nova Scotia	*Chairman*	Dr. A. S. Wenning
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	Secretary-Treasurer	Dr. R. B. Robertson
British Columbia	*Chairman*	Dr. H. S. Randall
	Secretary-Treasurer	Dr. H. B. Graves

BOOK REVIEWS

STELLATE GANGLION BLOCK. By DANIEL C. MOORE, M.D. Springfield, Ill.: Charles C. Thomas; Toronto: Ryerson Press. 1954. $11.50.

This well-illustrated monograph of 280 pages deals with all phases of stellate ganglion block. The original dissections of the thoraco-cervical portion of the sympathetic nervous system deserve special mention. In addition to this excellent section on the anatomy of the region and the distribution of the thoraco-cervical sympathetic elements, the author deals with the physiology of this portion of the sympathetic nervous system, agents which may be used for blocking it, various techniques, including the use of X-rays and radio opaque substances, in performing stellate ganglion block. The author has had wide experience of this technique, and has made an exhaustive review of the literature. His discussion of the indications for the use of stellate ganglion block are brief but to the point. This volume should certainly be read by every anaesthetist with an interest in regional anaesthesia, and the reviewer would hope that it will come to the attention of many of our colleagues in the profession who are unaware of the benefits which may be derived from block of the stellate ganglion as a therapeutic measure.

R.A.G.

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ANAESTHESIA IN OPHTHALMOLOGY. By WALTER S. ATKINSON, M.D. Springfield, Ill.: Charles C. Thomas, Toronto: Ryerson Press. 1955. $3.50.

In this 83-page monograph the author discusses mainly all phases of local anaesthesia for operations on the eye and orbit. The selection, preparation, and preoperative sedation of the patient are described in considerable detail, and several important points concerning the necessity of individual selection of sedation and psychological preparation of the patient for operation are emphasized by graphs and cartoons. There is a short reference to the use of Dramamine, Phenergan, and Thorazine for the control of postoperative nausea and vomiting.

Local anaesthesia is described under the headings of topical, infiltration and block techniques. The advantages and disadvantages of the various accepted local anaesthetic drugs are discussed. An outline of the anatomy of the eye is given and the various techniques are clearly described in great detail with the aid of many diagrams and photographs.

Several pages are devoted to the question of toxic reaction to the local anaesthetic drugs. The signs and symptoms are described and a very comprehensive discussion on the emergency treatment of severe toxic reactions is given. Some very good pen point cartoons add a graphic touch to this important point.

The question of general anaesthesia is dealt with rather briefly and in a general way. General anaesthesia is advocated for operations on children, for extensive operations for removal of orbital tumours, and for nervous people for enucleations, muscle operations etc. One reference is given for the use of Pentothal and Curare in intraocular surgery.

G.F.S.

SELECTION OF ANESTHESIA. By JOHN ADRIANI, M.D. Springfield, Ill.: Charles C. Thomas; Toronto: Ryerson Press. 327 pages. $7.25.

The surgeon and the internist are often faced with the problem not only of assessing and advising the practitioner and the patient regarding the outcome of an operation, but also of estimating the patient's suitability for an anaesthetic, or type of anaesthetic. This text has been written by Dr. Adriani especially for guidance under such circumstances, and does not deal with the technique of anesthetic administration.

Dr. Adriani is Director of the Department of Anesthesiology, Charity Hospital and Professor of Surgery, Tulane University, Louisiana. His book is well written and easy to read, with the chapters and paragraphs clearly headed up and indexed. He divides the subject into three main parts: (1) The Pharmacology and Clinical Application of Various Drugs in Common Use; (2) Various Diseases Complicating the Surgical Condition; (3) The Choice of Anesthesia in Relation to the Operation to be Performed.

The consultant will find this book most helpful and the anaesthetist and student will also profit greatly by reading it.

S.M.C.

CORRECTIONS

Volume 2, No. 1, January 1956. "Untoward Reactions to Succinylcholine," by JOHN I. DAVIES, M.D., F.R.C.P.(C), F.F.A.R.C.S., D.A.

On page 12, line 2, "75°C" should read "75° Fahrenheit."

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Volume 2, No. 2, April 1956. "Panel Discussion of Questions Submitted at the Closing Session of the Annual Meeting of the Canadian Anaesthetists' Society, June 23, 1955."

Page 148, Question 10 (Dr. Wyant). The third and fourth sentences should read: "It is one of the arguments of some surgeons that you cannot get as good muscle relaxation for abdominal operations with muscle relaxants as you can with subarachnoid spinal anaesthesia. Of course this is not true, but there are certain procedures in which spinal anaesthesia is certainly valuable."

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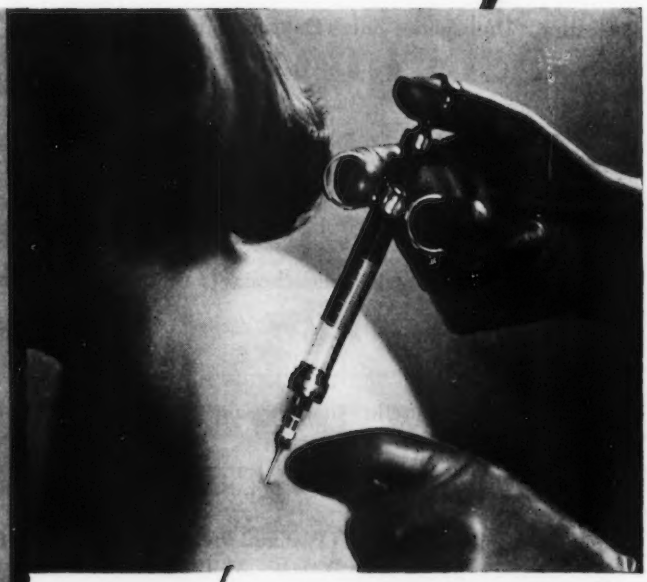

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